LOAD CHARACTERISTICS AND RESPONSES IN MALE ATHLETES WITH PATELLAR TENDINOPATHY

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ABSTRACT

Laura Stanley Pietrosimone: Loading Characteristics and Responses in Male Athletes with Patellar Tendinopathy
(Under the direction of Darin A. Padua)

Context: Clinical management of tendinopathy is difficult, as tendon pathoetiology occurs on a continuum with inconsistent associations between structural pathology and pain. Tendon is highly responsive to mechanical load; however, load mismanagement can trigger homeostatic imbalances that lead to the development of tendinopathy. There is a need to characterize loading profiles and responses of individuals along the tendinopathic continuum to inform improved clinical management strategies. **Objective**: To evaluate differences in biomechanical and loading volume profiles and to determine the effects of an acute bout of patellar tendon isometric loading exercise on lower extremity landing biomechanics in male athletes with patellar tendinopathy. **Participants**: 43 male athletes with and without unilateral patellar tendinopathy. **Interventions**: All participants (SYM, ASYM, CON) performed double- and single-limb jump-landing tasks and one-week physical activity monitoring period. Additionally, participants in the SYM and ASYM groups underwent a randomized cross-over protocol on two additional testing days to examine the acute effects of an isometric patellar tendon loading exercise protocol on landing biomechanics. **Main Outcome Measures**: Loading magnitude variables included tri-planar hip and knee kinematics, kinetics, and energetics for the involved limb. Load frequency and duration

variables included steps/day and amount of moderate-to-vigorous-physical activity/day, respectively. Load volume was estimated from load magnitude and frequency variables using validated equations. **Results**: SYM participants demonstrated patterns of under-loading, specifically in sagittal plane knee motion, patellar tendon force, and energy absorption during landing, and reduced load volume, compared to CON participants. There was no evidence of over-loading on any load magnitude, frequency, or duration variable for the ASYM participant. There was no acute effect of the isometric exercise protocol on any biomechanical measure for both the SYM and ASYM groups. **Conclusions**: Load magnitude, frequency, duration, and volume are all important metrics to measure and monitor in athletes at different stages of the continuum of patellar tendinopathy. Future research should evaluate the effects of isometric exercise protocols on movement profiles using longer duration exercise prescription and on individuals with higher magnitudes of tendon pain. Future research should continue to develop load monitoring strategies to improve tissue capacity and self-reported function in individuals with patellar tendinopathy.

DEDICATION

This dissertation is dedicated to two of my greatest role models:

Richard “Poppy” Joseph Crowder, Sr.

Richard “Uncle Rick” Joseph Crowder, Jr.

For the way they overcame obstacles. For their enthusiasm for life, both things big and small. For the gratitude with which they approached every day and every experience. For the way they put family before everything else. For their smiles, their laughs, and their six-foot-seven-inch bear hugs. For always strongly supporting their granddaughter and niece to pursue her goals. Poppy and Uncle Rick, we miss you dearly, but your legacy lives on in the eyes of the people fortunate enough to know you. In my personal and professional life, I pledge to strive to leave a legacy as meaningful as yours.

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To my husband, Brian, thank you for being by my side along this journey. You inspire me every day by your passion for your work, but most importantly by your love for family. You make me laugh. You make me better. I could not have travelled this road without you. It has been a true adventure, but I wouldn't want it any other way. I am so thankful that we are teammates for life and cannot wait for the many memories to come.

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amazing of a spouse, parent, colleague, and friend as both of you. I love you and am forever thankful for you.

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To my patients, past and future, thank you for trusting me, teaching me the power of what it can mean to help someone achieve their goals, and for inspiring me to be a leader. I pledge to

always ensure that my research will center around improving the quality of your lives in meaningful ways.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

ASYM: Asymptomatic

cF_{PT}: Cumulative Patellar Tendon Force

CON: Control

F_{PT}: Peak Patellar Tendon Force

$F_{PT}I$: Patellar Tendon Force Impulse

HSR: Heavy Slow Resistance

KEM: Knee Extension Moment

KEMI: Knee Extension Moment Impulse

KP: Knee Power

KW: Negative Knee Work

NRS: Numeric Rating Scale

PT: Patellar Tendinopathy

PTA: Patellar Tendon Abnormality

SYM: Symptomatic

US: Ultrasonography

UTC: Ultrasound Tissue Characterization

vGRF: Vertical Ground Reaction Force

CHAPTER 1: INTRODUCTION

Patellar tendinopathy is prevalent in individuals who are physically active, particularly athletes who participate in sports with repetitive jumping manueveurs.[1–5] While some athletes are able to maintain sport participation, the long-term consequences of chronic tendinopathy include reduced physical activity and quality of life,[6,7] with up to 53% of individuals with symptomatic patellar tendinopathy quitting their sport due to chronic tendon pain.[8] Clinical management of tendinopathy is difficult, as tendon pathoetiology occurs on a continuum with inconsistent associations between structural pathology and pain.[9–11] Tendon is highly responsive to mechanical load; however, load mismanagement can trigger homeostatic imbalances that lead to the development of structural pathology and/or symptoms.[10] While laboratory-based assessments have established some evidence of altered biomechanics in adults with a history of patellar tendinopathy, there is a lack of literature directly comparing biomechanical movement profiles of symptomatic and asymptomatic individuals with patellar tendon structural pathology. Furthermore, the laboratory environment cannot account for the influence of cumulative external load incurred during real-world physical activity on variables associated with the development of patellar tendinopathy.

Therefore, despite strong evidence of the load-response characteristics of tendon,[10,12] there is a ***critical gap*** in our knowledge of how cumulative external load influences the development and progression of tendinopathy. Filling this gap would give rise to ***innovative controlled load management strategies*** that aim to mitigate the progression of tendon pathology

and consequent reduction in physical activity. Additionally, while eccentric-based strength-training protocols have traditionally been utilized in the treatment of chronic tendinopathies,[13–15] emerging evidence supports the use of isometric exercise for individuals with symptomatic patellar tendinopathy. Isometric exercise has recently been shown to improve pain and self-reported function in adults with patellar tendinopathy,[16–18] resulting in excellent patient compliance and tolerance when implemented in-season.[16] However, the acute effects of isometric patellar tendon loading on landing biomechanics is unknown. Determining the effects of this novel exercise intervention on movement characteristics in clinical populations may allow for improved subgrouping of patients into impairment-based rehabilitation programs and subsequently improve clinical effectiveness.

The ***overall objective*** of this study is to determine the effects of an acute bout of patellar tendon isometric loading exercise on lower extremity landing biomechanics, and to evaluate differences in biomechanical profiles of individuals at varying stages of the tendon pathology continuum using both laboratory and real-world movement assessments. Our ***approach*** will utilize a randomized cross-over study design to assess acute intervention effects on lower extremity kinetic and kinematic biomechanical variables during landing, and a cross-sectional quantification of one-week cumulative external load using wearable technology.

Aim 1. To ascertain the impact of symptomatic PTA and asymptomatic PTA on lower extremity landing kinematics and kinetics.

Aim 2. To ascertain the impact of symptomatic PTA and asymptomatic PTA on cumulative external load during a one-week monitoring period.

Aim 3. To investigate whether an acute isometric patellar tendon loading exercise protocol changes lower extremity landing kinematics and kinetics in individuals with symptomatic and asymptomatic PTA.

The proposed project is ***innovative*** because it will be the first to establish the effects of patellar-tendon specific loading exercise on biomechanical movement profiles of individuals along the continuum of tendon pathology, and the first to monitor cumulative external load in a tendinopathic population. Long-term, an efficacious real-world monitoring system will enhance clinical practice by allowing for timely identification of trends in loading that may influence the development of structural pathology and altered biomechanics in multiple patient populations.

Specific Aims, Research Question Objectives, & Hypotheses

Specific Aim 1. To ascertain the impact of symptomatic PTA and asymptomatic PTA on lower extremity landing kinematics and kinetics.

Research Questions

1.1 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different *sagittal and frontal plane knee and hip joint angles* during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?

1.2 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate *different internal sagittal and frontal plane knee and hip joint moments* during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?

1.3 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different *peak vertical ground reaction force magnitudes and vertical ground reaction force*

loading rates during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?

1.4 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different *peak patellar tendon force magnitudes and patellar tendon force loading rates* during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?

1.5 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate differences in *inter-limb symmetry for kinetic variables* during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?

Hypotheses

Hypothesis 1: Individuals with symptomatic PTA and asymptomatic PTA will demonstrate different lower extremity landing kinematics and kinetics compared to healthy controls.

1.1 Individuals with symptomatic PTA will demonstrate lesser sagittal plane knee and hip flexion displacement on the involved limb, while individuals with asymptomatic PTA will demonstrate greater sagittal plane knee and hip flexion displacement on the involved limb compared to the matched limb of healthy controls.

1.2 Individuals with symptomatic PTA will demonstrate lesser net sagittal plane knee and hip internal extension moment on the involved limb, while individuals with asymptomatic PTA will demonstrate greater net sagittal plane knee and hip internal

extension moment on the involved limb compared to the matched limb of healthy controls.

1.3 Individuals with symptomatic PTA will demonstrate lesser peak vertical ground reaction force on the involved limb, while individuals with asymptomatic PTA will demonstrate greater peak vertical ground reaction force on the involved limb compared to the matched limb of healthy controls.

1.4 Individuals with symptomatic PTA will demonstrate lesser peak patellar tendon force on the involved limb, while individuals with asymptomatic PTA will demonstrate greater peak patellar tendon force on the involved limb compared to the matched limb of healthy controls.

1.5 Individuals with symptomatic PTA will demonstrate greater magnitude inter-limb asymmetry in kinetic variables during the loading phase of the landing tasks compared to individuals with asymptomatic PTA and healthy controls.

Rationale

Previous research has investigated biomechanical profiles of individuals with either structural or symptomatic evidence of patellar tendinopathy when performing sport-specific tasks, such as jumping and landing.[19–24] However, the volume of published research in the area of biomechanical profiles of individuals with patellar tendinopathy is much smaller than that of other patient populations, such as individuals following anterior cruciate ligament injury. Previous studies have demonstrated differences in lower extremity kinematics, kinetics, and energetics between individuals with and without patellar tendinopathy symptoms,[19–22] as well as between asymptomatic individuals with and without structural abnormalities.[23,24] However, there are no studies to-date that have compared biomechanical characteristics that control for both

patellar tendon structure and tendinopathy symptomology, and simultaneously include a healthy control group (no structural pathology or symptomology). This study is the first to compare biomechanical profiles of three distinct groups: individuals who are asymptomatic with a PTA, symptomatic with a PTA, and asymptomatic without PTA (health control). Through this design, we will be able to assess the independent effects of both structural pathology and pain on biomechanical profiles during sport-specific tasks. The clinical relevance of this design is that strives to determine if biomechanical profiles are different between individuals at differing stages along the continuum of tendon pathology, which could inform the development of enhanced impairment-based, individualized treatment programs.

Target Journal

The manuscript reporting the results of this specific aim will be prepared for submission to *Medicine & Science in Sports & Exercise (MSSE)* (Impact Factor: 4.041). Previous studies reporting biomechanical profiles of tendinopathic populations have been published in *MSSE,*[23,25,26] so the results of our study, which uniquely controls for both structural and symptomatic deficits, will provide an important continuation of work that has been selected by *MSSE* for publication in the past.

Table 1.1. Summary of Aim 1

Research Question Objectives	***Dependent Variables***	***Statistical Analysis***
	The following DVs will be assessed for involved PTA limbs and matched healthy control limb.	
1.1 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different sagittal and frontal plane knee and hip joint angles during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?	Knee flexion angle Knee valgus angle Hip flexion angle Hip adduction angle	95% confidence interval waveform comparisons
1.2 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different internal sagittal and frontal plane knee and hip joint moments during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?	Internal knee extension moment Internal knee abduction moment Internal hip flexion moment Internal hip adduction moment	95% confidence interval waveform comparisons
1.3 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different peak vertical ground reaction force magnitudes and vertical ground reaction force loading rates during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?	Peak vGRF	95% confidence interval waveform comparisons
1.4 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different peak patellar tendon force magnitudes and patellar tendon force loading rates during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?	Peak patellar tendon force (F_{PT}) Patellar tendon force impulse	95% confidence interval waveform comparisons
1.5 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate differences in inter-limb symmetry for kinetic variables during the loading phase of each landing task compared to individuals who are asymptomatic and without PTA (healthy control group)?	Internal knee extension moment Vertical ground reaction force Patellar tendon force	95% confidence interval waveform comparisons

Specific Aim 2. To ascertain the impact of symptomatic PTA and asymptomatic PTA on cumulative external load during a one-week monitoring period.

Research Questions

2.1 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different average steps-per-day during a one-week monitoring period compared to individuals who are asymptomatic and without PTA (healthy control group)?

2.2 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different cumulative patellar tendon loads compared to individuals who are asymptomatic and without PTA (healthy control group)?

Hypotheses

Hypothesis 2: Individuals with symptomatic PTA and with asymptomatic PTA will demonstrate different average daily and cumulative loading volume during a one-week monitoring period than healthy controls, such that:

2.1 Individuals with symptomatic PTA will demonstrate less loading volume than both individuals with asymptomatic PTA and healthy controls.

2.2 Individuals with asymptomatic PTA will demonstrate greater cumulative patellar tendon loads than both individuals with symptomatic PTA and healthy controls.

Rationale

Musculoskeletal injury is a primary factor related to reduced participation in physical activity, which can result in numerous negative health outcomes.[27,28] Overuse injuries, such as tendinopathy, typically result from the mismanagement of load over time. As a mechano-responsive tissue, normal tendon readily adapts to loading with degradation and synthesis of tendon matrix, and previous research has demonstrated that tendon can respond favorably to

controlled loading in certain athletic populations.[29–31] However, loading frequency, intensity, and duration may influence tendon response, particularly in the presence of maladaptation (structural pathology and/or symptomology).[10] Therefore, quantification of key objective physical activity characteristics, as outlined by the FITT principle[32,33] may provide critical information on tendon adaptation to load that is currently not systematically evaluated in standard clinical practice around patellar tendinopathy.

A limitation of traditional biomechanical assessments in the area of musculoskeletal injury and sports medicine is the exclusive use of laboratory measures (i.e. three-dimensional motion capture) to study associations between movement characteristics and injury. While considered the gold-standard movement assessment tool, three-dimensional motion capture only provides a brief, controlled snapshot of an individual's biomechanical profile, which does not account for cumulative loading repetition or the influence of overall physical activity exposure. Therefore, real-world physical activity monitoring is a critical missing piece in the study of overuse injury development. Previous literature has demonstrated associations between high training and competition workloads and injury.[34,35] Specifically, high training load volume (training hours/week, match/week) increases the risk of patellar tendinopathy in adolescent male and female volleyball athletes (OR: 1.72-3.38).[36] Additionally, in a cohort of collegiate female volleyball athletes, cumulative season training load was negatively association with post-season VISA-P scores (r=-0.512, p=0.043), indicating that athletes with higher loads during the competitive season reported more post-season patellar tendon pain. (Stanley et al, in progress)

The utility of quantifying physical activity outcomes in this population is to obtain a more objective understanding of the associations between cumulative external load and clinical manifestations of patellar tendinopathy (i.e. structural pathology and pain). Recent studies have

quantified physical activity metrics in various pathologic populations. Individuals with chronic ankle instability were found to participate in 24 less minutes of physical activity per day than healthy counterparts[37], and individuals following ACLR took on average 2000 less steps-per-day than healthy controls.[38] In a population of adults with osteoarthritis (OA), combined laboratory- and real-world based knee joint loading metrics were better able to distinguish between individuals with and without OA than traditional laboratory-based assessments alone.[39] Therefore, based on the foundational knowledge of tendon as a mechano-responsive tissue, and growing evidence of altered real-world loading in pathological populations, measuring and monitoring real-world loading metrics is a critical component in the study of overuse injuries.

Target Journal

The manuscript reporting the results of this specific aim will be prepared for submission to the *American Journal of Sports Medicine (AJSM)* (Impact Factor: 4.517). There is a growing interest among sports medicine professionals to utilize wearable sensor technology for pre-injury prevention to monitor athletes following injury. Therefore, we feel that the results of this study, which will be the first to-date to investigate real-world loading difference in individuals with varying stages of patellar tendinopathy, fit with the mission of *AJSM* to be a leading journal in sports medicine. *AJSM* has also recently published a study[38] investigating real-world loading comparisons between un-injured controls and individuals following anterior cruciate ligament reconstruction (ACLR), indicating that this area of patient-management is of interest to their editors and readership.

Table 1.2. Summary of Aim 2

Research Question Objectives	*Dependent Variables*	*Statistical Analysis*
2.1 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different average steps-per-day during a one-week monitoring period compared to individuals who are asymptomatic and without PTA (healthy control group)?	Average steps-per-day Average MVPA-per-day	One-way ANOVA
2.2 Do individuals with symptomatic PTA and asymptomatic PTA demonstrate different cumulative patellar tendon loads compared to individuals who are asymptomatic and without PTA (healthy control group)?	Cumulative F_{PT}	One-way ANOVA

Specific Aim 3. To investigate whether an acute isometric patellar tendon loading exercise protocol changes lower extremity landing kinematics and kinetics in individuals with symptomatic and asymptomatic PTA.

Research Questions

3.1 Does an acute isometric patellar tendon loading exercise protocol change *sagittal and frontal plane knee and hip joint angles* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to those with asymptomatic PTA?

3.2 Does an acute isometric patellar tendon loading exercise protocol change *net internal sagittal and frontal plane knee and hip joint moments* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to the asymptomatic PTA?

3.3 Does an acute isometric patellar tendon loading exercise protocol change *peak vertical ground reaction* force during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to the asymptomatic PTA?

3.4 Does an acute isometric patellar tendon loading exercise protocol change *peak patellar tendon force* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to the asymptomatic PTA?

Hypotheses

Hypothesis 3. The acute isometric patellar tendon loading exercise protocol will elicit different changes in lower extremity landing kinematics and kinetics in individuals with symptomatic PTA compared to the asymptomatic PTA.

3.1 The acute isometric patellar tendon loading exercise protocol will increase sagittal plane knee and hip joint angles in the symptomatic PTA group, relative to the asymptomatic PTA.

3.2 The acute isometric patellar tendon loading exercise protocol will alter sagittal plane knee and hip joint moments, specifically greater internal knee extension moment and lesser internal hip flexion moment, in the symptomatic PTA group, relative to the asymptomatic PTA.

3.3 The acute isometric patellar tendon loading exercise protocol will increase peak vertical ground reaction force in the symptomatic PTA group relative to the asymptomatic PTA.

3.4 The acute isometric patellar tendon loading exercise protocol will increase peak patellar tendon force in the symptomatic PTA group relative to the asymptomatic PTA.

Rationale

Approximately 20-30% of asymptomatic athletes demonstrate patellar tendon structural abnormalities.[1,40–42] Furthermore, there is an approximate four-fold increased risk of symptom development in individuals with a patellar tendon abnormality.[43] Due to the prevalence of structural- and symptom-based impairments in athletes participating in sports marked by high frequency and intensity loading on the knee joint, determining the effectiveness of targeted

treatment strategies is critical. Tendon is a visco-elastic tissue that readily responds to loading via mechanotransduction processes;[12] therefore, using exercise-based therapies to promote positive adaptation when tendon is structurally compromised both before and after symptom onset is supported. Isometric patellar tendon loading exercise has recently been shown to both acutely and chronically decrease tendon pain, improve quadriceps strength, and improve self-reported knee function during sport in individuals with symptomatic patellar tendinopathy.[16–18]

However, the effects of this targeted tissue-specific loading protocol on lower extremity biomechanics has not yet been investigated. Athletes with symptomatic patellar tendinopathy demonstrate load avoidance movement strategies during sport-specific tasks, including reductions in sagittal plane knee displacement and mechanical energy absorption, lesser vertical ground reaction force, and lesser internal knee extension moment.[19,21,22] This study will be the first to test the acute effects of an isometric patellar tendon loading exercise protocol[17] on landing biomechanics in individuals across the tendon pathology continuum (both asymptomatic and symptomatic individuals with structural abnormalities). Using isometric loading interventions to acutely change movement biomechanics may provide an important next step in rehabilitation paradigms for tendinopathy as a method to promote improve load-tolerance during and stimulate positive mechano-transductive responses in individuals with tendon pathology.

Target Journal

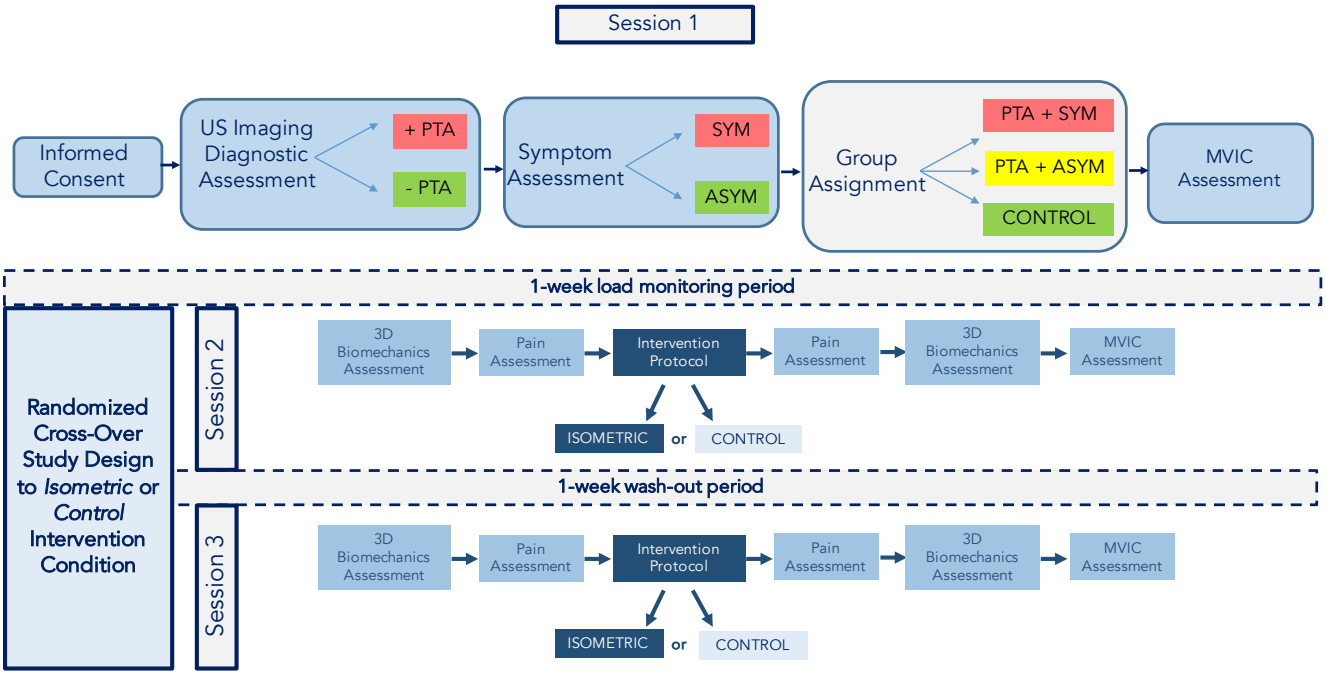
The manuscript reporting the results of this specific aim will be prepared for submission to the *Journal of Orthopedics and Sports Physical Therapy (JOSPT)* (Impact Factor: 2.55). Tissue-specific loading interventions are common in clinical practice for the treatment of tendinopathies. Therefore, the results of this specific aim have the potential to aid in the advancement of therapeutic interventions to treat individuals with patellar tendinopathy and are

appropriate for *JOSPT*, as this journal seeks to publish clinically-relevant studies specific to common orthopedic conditions. Additionally, this journal is housed within my professional organization, the American Physical Therapy Association (APTA).

Table 1.3. Summary of Aim 3

Research Question Objectives	***Dependent Variables***	***Statistical Analysis***
3.1 Does an acute isometric patellar tendon loading exercise protocol change *sagittal and frontal plane knee and hip joint angles* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to those with asymptomatic PTA?	The following kinematic variables will be calculated for initial contact, peak, and displacement across the loading phase of the landing task for the involved PTA limb: Knee flexion angle Knee valgus angle Hip flexion angle Hip adduction angle	Between groups: 2x2 mixed-model repeated-measures analysis of variance ANOVA on change scores for involved limbs from pre- to post-intervention
3.2 Does an acute isometric patellar tendon loading exercise protocol change *net internal sagittal and frontal plane knee and hip joint moments* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to those with asymptomatic PTA?	The following kinetic variables will be calculated across the descending phase of the landing task for the involved PTA limb: Peak Internal knee extension moment Peak Internal knee abduction moment Peak Internal hip flexion moment Peak Internal hip adduction moment	Between groups: 2x2 mixed-model repeated-measures analysis of variance ANOVA on change scores for involved limbs from pre- to post-intervention
3.3 Does an acute isometric patellar tendon loading exercise protocol change *peak vertical ground reaction force* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to those with asymptomatic PTA?	Peak vGRF	a. 2x2 mixed-model repeated measures ANOVA on change scores for involved limbs peak vGRF from pre- to post-intervention
3.4 Does an acute isometric patellar tendon loading exercise protocol change *peak patellar tendon force* during the loading phase of the double-limb landing task in individuals with symptomatic PTA compared to those with asymptomatic PTA?	Peak patellar tendon force (F_{PT}) Patellar tendon force impulse	a. 2x2 mixed- model repeated measures ANOVA on change scores for involved limbs peak F_{PT} and F_{PT} impulse from pre- to post-intervention

Figure 1.1. Overview of the proposed study methodology to assess the acute effects an isometric patellar tendon loading exercise protocol on lower extremity landing biomechanics, and to quantify cumulative external load metrics over a one-week training period.



Independent Variable

1. Patellar Tendon Structural and Symptom Profile
    a. *Symptomatic / PTA vs. Asymptomatic / PTA vs. Asymptomatic / No PTA*

Dependent Variables

1. Biomechanical Variable Change Scores following the Isometric Patellar Tendon Loading Exercise Intervention collected over the loading phase of the double-limb landing task; Baseline Biomechanical Variables 95% Confidence Interval Waveform Comparisons over the entire stance phase of the double-limb landing task:
        i. Sagittal plane knee joint angle
        ii. Frontal plane knee joint angle
        iii. Sagittal plane hip joint angle
        iv. Frontal plane hip joint angle
        v. Net internal sagittal plane knee joint moment
        vi. Net internal frontal plane knee joint moment
        vii. Net internal sagittal plane hip joint moment
        viii. Net internal frontal plane hip joint moment
        ix. Vertical ground reaction force
        x. Patellar tendon force
        xi. Patellar tendon force impulse
2. Cumulative External Load Variables from 1-Week Load Monitoring Period
    a. Average daily steps-per-day
    b. Average moderate-to-vigorous-physical-activity per day
    c. Cumulative patellar tendon force estimation (cF_{PT})

CHAPTER 2: REVIEW OF LITERATURE

The Landscape of Musculoskeletal Injury in Youth Sports

The last half-century has witnessed a steady rise in sports participation among youth athletes.[44] In the United States, high school sports participation increased by approximately 80% between 1971 and 2005,[44] which has been attributed to growing opportunities for females in sport and the growing emphasis on health promotion in youth.[45,46] In a large cross-sectional, nationally representative sample, approximately 62% of high school students (70% of males; 53% of females) reported participating in at least one sport.[47] Nearly 8 million boys and girls participated in organized high school sports during the 2014-2015 school year.[48] The importance of physical activity in youth populations has been recognized by national and international governing bodies as a priority from health behavior and economic perspectives. The World Health Organization recommends at least sixty-minutes of moderate to vigorous physical activity (MVPA) per day for youths aged 5-17, and notes that greater than sixty minutes-per-day may provide additional health benefits.[49] Physical activity and sports participation in the youth population has been associated with numerous positive health and social behaviors[47] and continued physical activity into adulthood,[46] lowering the risk of a variety of disease conditions, including cardiovascular disease, diabetes, and various cancers.[50]

Despite the well-accepted benefits of promoting physical activity in youth populations, an unintended consequence of rising youth sports participation is the increased potential for sports-related injury. Epidemiologic data demonstrates an overall increase in pediatric and

adolescent sports-, recreation-, and exercise-related (SRE) injuries, due to both acute and chronic mechanisms.[51] During the 2014-2015 school year, high school athletes in the United States suffered an estimated 1.2 million injuries.[52]

Of particular concern is the high prevalence of musculoskeletal (MSK) injury in youth athletes. MSK injury diagnoses constitute the majority of self-reported SRE injuries in both high school and collegiate populations.[53–55] In a large-scale national survey, 64% of reported sport-related injury episodes occurred among individuals aged 5-24, and were reported as approximately 41% higher than national estimates of injuries that require emergency department visits.[53] Additionally, the majority of reported SRE injuries were to the upper (31.2%) and lower (38.9%) extremities.[53] The consequences of MSK injuries sustained at a young age are numerous, including economic, social, and long-term outcomes. In the United States, MSK SRE injuries comprise up to 64% of all emergency department visits by individuals 19 years old and younger.[56] While there is limited data describing long-term health impacts of injuries sustained in young athletes, this is an area gaining increasing interest due to the rise in youth sport participation. In fact, recent evidence demonstrates that athletes with a history of MSK injury or a current MSK injury score lower than uninjured counterparts on validated quality of life measures, including health problems and social functioning, and report less perceived physical capability.[57–60]

While MSK injuries sustained through acute, traumatic mechanisms, such as anterior cruciate ligament (ACL) injury, are debilitating, costly, and result in time loss for sport participation,[61–63] chronic, overuse MSK injuries are often equally as challenging and burdensome on both healthcare practitioners and patients alike. Most overuse injuries have a multi-factorial pathoetiology, rendering difficult diagnosis and treatment pathways. Patellar

tendinopathy, a common overuse injury in athletes, is a challenging condition to treat due to its varied clinical presentation, specifically the inconsistently-present clinical indicators of pain and structural pathology. Tendinopathies result from mismanagement of external load, typically in the direction of tissue overloading.[9,64] Traditional management of lower limb tendinopathies emphasized notable reductions in external load until full resolution of symptoms was achieved.[65] Eccentric exercise protocols, particularly for chronically symptomatic tendinopathies, constitute the common standard of care for tendinopathy,[14,66] and are likely one of the most widely-spread implemented treatment paradigms in rehabilitative musculoskeletal clinical practice. However, evidence supporting a continuum of tendon pathology, described by progressive stages of structural pathology, suggests that one-size-fits-all eccentric exercise protocols may not be appropriate to prescribe for all stages of tendinopathy.[10] Emerging evidence demonstrates positive effectiveness of isometric loading exercise protocol on pain and self-reported function in athletes with patellar tendinopathy.[16–18] However, to-date there is no literature describing the effects of an acute patellar tendon loading exercise protocol on lower extremity biomechanics, despite demonstrated differences in lower extremity biomechanics between individuals with and without patellar tendinopathy symptoms,[19–22] as well as between asymptomatic individuals with and without structural abnormalities.[23,24] Finally, advances in wearable technology allow for quantification of physical activity metrics that provide objective insight into cumulative external loading. Recent evidence demonstrates that individuals with a history of knee injury participate in less physical activity than to healthy, uninjured peers.[38] It is likely that the quantification of cumulative physical activity is a critical piece missing from current investigations of factors related to the development and treatment of tendinopathy, and is an area that should be explored based on the well-accepted load-responsiveness of tendon.

This review will focus on three primary areas around the topic of patellar tendinopathy to support the current study: 1) epidemiology and pathoetiology, 2) intrinsic and extrinsic factors, and 3) exercise-based intervention paradigms. Overall, the aims of this study seek to contribute to the current understanding of the continuum of tendon pathology,[10] and inform our understanding of modifiable factors that can be directly applied to the clinical management of tendinopathy.

SECTION 1: Injury Epidemiology & Pathoetiology

Epidemiology of Patellar Tendinopathy

Patellar tendinopathy is a chronic, overuse injury condition resulting from excessive tissue load.[67,68] Epidemiological studies have demonstrated a 2.5 – 14.4% prevalence of patellar tendinopathy in a diverse group of sports requiring high loading rates and power demands.[4,5] In particular, individuals participating in sports involving repetitive jumping and landing have been shown have the highest rates of patellar tendinopathy, due to the repetitive load placed on the tendon tissue. The prevalence in elite and recreational adult basketball athletes has been reported to be as high as 32% and 12%, respectively; a similar prevalence has been noted in elite (45%) and recreational (14%) volleyball athletes.[4,5] Athletes participating in other sports, such as track and field (running and high/long jump athletes), tennis, and skiing, have also been shown to readily develop tendinopathy.[67]

Sex differences in the incidence of patellar tendinopathy have been highlighted in numerous epidemiological studies. In both adolescent[36] and adult[69] athlete populations, males have been reported to develop PT more readily than females. In a cross-sectional multi-sport investigation of elite adult athletes, the prevalence of current patellar tendinopathy was significantly higher among males (13.5%) than females (5.6%).[5] Though not as thoroughly

studied as adult populations, an approximate 7% prevalence of patellar tendinopathy in adolescent (ages 14-18) athletes has been noted.[1] Moreover, in young elite volleyball athletes, boys have been shown to have four-times higher risk of developing PT than girls, independent of training and match exposure.[36] While the sex discrepancy appears to be consistent in different age groups and sports, due to increased sport participation among young females,[46] continued evaluation of both sexes should be pursued. For example, a high prevalence (26.6%) of anterior knee pain has been documented in female adolescent athletes assessed over three-years during pre-participation screenings, including higher prevalence in high-school (34.4%) versus middle-school (23.5%) aged athletes,[70] suggesting that young females should be monitored closely during sport participation. Additionally, as athlete's progress from junior to senior sporting levels, the incidence of patellar tendinopathy increases,[71] likely due to the cumulative chronic load from aggregated years of sport participation. Moreover, Hall et al (2015) demonstrated an increased risk of patellar tendinopathy in youth athletes who specialize in a single sport (OR: 1.27 – 4.0).[72]

Unlike an acute traumatic knee injury, the onset and progression of patellar tendinopathy does not typically result in immediately removal from sport. However, its progression and lack of resolution over time can lead to reduced capacity for sports participation at an athletes' normal frequency and intensity. Cook et al. (1997) demonstrated that over one-third of athletes who present with symptomatic patellar tendinopathy are unable to return to sport within six months.[7] Additionally, approximately 50% of athletes who develop recalcitrant patellar tendinopathy retire from sport due to the condition.[5] Like many chronic musculoskeletal conditions, persistent tendinopathy often results in an overall reduction in physical activity[6], which may initiate a cascade of secondary negative health consequences over a lifespan. Due to the complex

pathoetiology of tendinopathy, however, it is thought that the effects of chronic tendinopathy on performance, self-perceived function, and quality of life are likely underestimated.[36]

The prevalence of patellar tendinopathy in youth athletes, particularly in those who participate in sports involving high frequency and intensity of repetitive jumping and landing, and are exposed to high cumulative training volumes, necessitates continued attention from the sports medicine clinical and research communities. This may be of particular importance during a period when youth sports participation is increasing exponentially in the United States, not only to reduce onset and progression of pathology, but to also ensure that young individuals are able to maintain healthy levels of physical activity as they mature. Knowledge of risk factors for patellar tendinopathy provides avenues to improve treatment strategies to decrease the burden of this condition in young athletes, which in turn will promote lifelong physical activity.

Models of Tendon Pathoetiology

The management of tendinopathy in clinical settings can be challenging, due largely to the varied clinical presentation, specifically inconsistent relationships between tendon structural pathology, function, and pain.[10] Multiple models are represented in existing literature that outline the pathoetiology of tendinopathy. These models all suggest that the development of tendinopathy occurs along a continuum, but that distinct factors initiate the pathoetiologic cascade. Specifically, collagen matrix disruption, inflammation, and tendon cell response have been identified in previous literature as key factors driving tendinopathic processes.[73]

One of the earliest models suggested that tendinopathy initially develops from the occult disruption of the tendon collagen matrix.[73] Disruption and/or tearing of the collagen matrix is thought to precede the onset of pain; subsequently, the inability of disrupted tendon collagen matrix to perform its normal mechano-transductive role leads to under-loading and degenerative

pathology.[73,74] This model suggests that degenerative pathology is irreversible, and is considered end-stage pathology. A limitation to this model is that it does not consistently describe phases of tendon adaptation that may precede degeneration, which is a critical facet when identifying injury prevention strategies in high-risk population.

Further, inflammatory models are aligned around the notion that inflammatory substances drive tendinopathic processes. While widely accepted amongst clinical and scientific groups, there are several limitations to this model. One of the key limitations is the lack of cellular inflammation that is present in pathological tendons. For decades, it was thought that the primary source of pain in tendinopathy was due to inflammation within the tendon (i.e. 'tendinitis'). However, numerous studies have clearly demonstrated that there is a lack of intra-tendinous inflammation present in tendinopathy. Two key investigations conducted by Khan et al (1996) and Sanchis-Alfonso (2001) provide strong evidence to support this shift.[75,76] In both of these studies, biopsies of patellar tendons from individuals with recalcitrant symptoms were taken at the time of surgery. Histochemical analysis indicated consistent changes to the structural matrix, including poorly aligned irregular collagen fibers, shift from Type I to Type III collagen, increased swollen tenocytes, fibrils, and fluid, and heightened expression of matrix-degrading cytokines (i.e. TNF-alpha). Interestingly, there was a complete lack of inflammatory cells and biomarkers. Instead, both studies noted the presence of neuronal-sprouting and increased vascularity within the pathologic tendon. Other studies have reported similar findings, specifically that neovascularization is commonly present in pathologic, symptomatic tendons.[76] Sanchis-Alfonso (2001) proposed that the pain experienced in tendon pathology is likely not from intra-tendinous inflammation, but instead from the heightened neovascularization that occurs in an attempt to repair and remodel over-stressed tendon.[76]

In summary, early models of tendon pathology were defined by two distinct classifications of tendon status: tendinitis (acute) and degenerative tendinosis (chronic).[65] The distinction between these two classifications was based largely on duration of symptoms and key aspects of the clinical exam. Specifically, tendinitis was classified based on the following key constructs:

- Acute onset with short duration of symptoms
- Inflammatory processes within the tendon proper (-itis), resulting in pain
- Lack of obvious structural changes to the extracellular matrix (ECM)
- Notable tenderness to palpation and observable/palpable focal swelling on physical exam

Degenerative tendon pathology (tendinosis) was classified based on the following key constructs:

- Chronic, recalcitrant symptomology (>6+ months)
- Notable changes to the extra-cellular matrix, including collagen disorganization or a complete loss of matrix integrity
- Irreversible structural changes that are unable to respond to load-based treatments
- +/- tenderness to palpation and observable/palpable focal swelling on physical exam

The Continuum of Tendon Pathology

Current evidence describing the pathoetiology of tendinopathy has evolved from these previous models, such that tendon pathology is now commonly described to occur over a continuum, from early reactive stages (acute) to later stages of dysrepair and degeneration (chronic).[9] In the context of clinical management of tendinopathy, treatments are most effective when they are tailored to match the stage of tendinopathy, as there is evidence that tendons at

different stages of the pathologic continuum may require different treatment approaches.[10] In fact, the prescription of inappropriate interventions to a tendon may lead to a worse outcome.[77]

Therefore, a shift in the model of tendon pathology to that of a continuum model has been largely supported over the last decade.[9,10] This model defines tendon pathology across three continuous stages, and contends that the movement from one stage to another is largely determined by imposed or external loading stimulus. There is considerable evidence demonstrating that external load is one of the primary etiological factors that influences tendon structural properties.[9,78,79] Biological tissue homeostasis has been described by Dr. Scott Dye (1996) as the *envelope of function,* or "the range of load that can be applied across an individual tissue in a given period without supraphysiologic overload or structural failure."[64] Tendon health is intimately related to mechanical homeostasis. Tendon adaptation occurs through mechanotransduction, the physiological process by which the body translates mechanical load into a cellular response that leads to structural change.[12] (see Section 3) Evidence describing structural changes that occur when a tendon is mechanically stimulated, or increasing the capacity of the tendon,[80,81] supports the continuum model that tendon pathology does develop along a continuum, without discrete "onset" and "resolution" points. Since external loading is a modifiable construct, understanding how tendon responds to both acute and cumulative loading, particularly in athletes at an elevated risk of developing overuse injuries, may improve tailored intervention delivery focused on load-based management.

The continuum model contends that load is not necessarily deleterious, but that the proper dosage and monitoring of load that is anabolic versus catabolic to tendon health is critical. The modification of load in the presence of pain is essential in order to promote positive versus negative adaptation, and to avoid a cascade of pathophysiological consequences[82] that amplify

the difficulty of clinical management. The three stages described by Cook et al. in the continuum model are: reactive, dysrepair, and degenerative tendinopathic stages (Table 2.1).

Table 2.1. Characteristics of stages of tendon pathology described in the continuum model from Cook et al (2009).[9]

	Proposed Etiology	**Histological Features**	**Clinical Features**	**Imaging Features**
Reactive Tendinopathy	Acute spike in tensile and/or compressive load, or direct blow to tendon	Proliferation of cells & matrix, changing shape of cells (↑ chondroid, thickening) Tendon swelling due to increased bound water to proteoglycans.	Acute onset following spike in external loading Initially diffuse anterior knee pain	Fusiform swelling Diffuse hypoechogenicity on US
Tendon Dysrepair	Continued external overload	Progressive matrix breakdown Increased cell production, including chondrocytic cells Increased proteoglycan production leading to collagen separation and matrix disorganization	Chronically overloaded Pain more localized	Matrix and collagen disorganization (wavier pattern) Fusiform swelling Increase areas of focal hypoechogenicity Small increases in neovascularization
Degenerative Tendinopathy	Chronic external overload	Notable matrix and cellular changes/heterogeneity, including areas of cell death (acellularity) Proliferation of neovascularization	Focal tendon swelling and pain History of repeated bouts of tendon pain	Distinct hypoechoic regions on US Large vessels on Doppler US High intratendinous signal on MRI

In the *reactive* stage**,** if a temporary alteration in external load (decrease in magnitude, frequency, or duration) is made, a tendon in this stage has the ability to positively adapt, becoming stronger (Wolff's Law) via remodeling and repair, and remain a functional, non-pathologic, non-symptomatic tendon. In the *dysrepair* stage, the modification of loading parameters can still be effective at allowing the tendon to recover and move back towards the reactive and normal stages. If a tendon in the dysrepair stages is continually loaded above

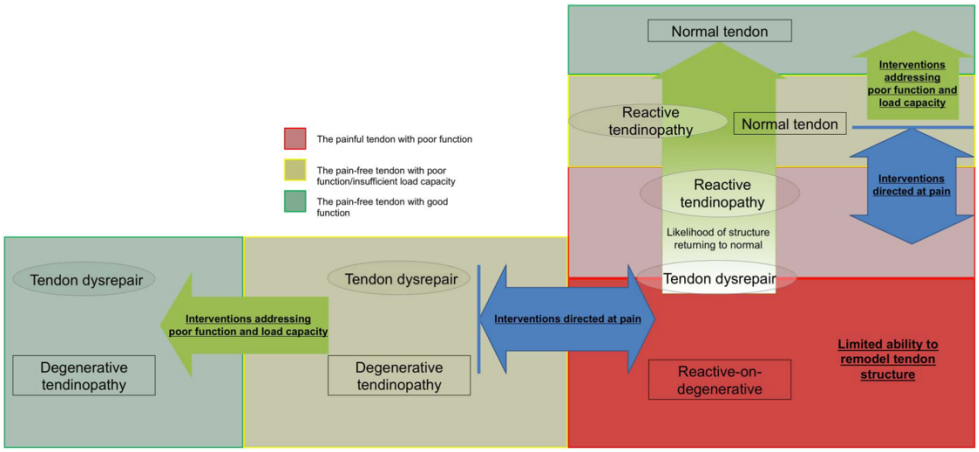
optimal levels, a catabolic pathway is promoted, stress-shielding occurs, and, over time, it is more likely to enter a degenerative stage. However, once in the *degenerative* stage**,** continued overload to the tendon has become catabolic, as the magnitude/frequency/duration of load has exceeded tendon's tolerance and allowed inadequate time to recover, remodel, and repair. There is an approximate four-fold increased risk of symptom development in individuals with a patellar tendon abnormality.[43] Evidence suggests that there is little capacity for tendon structure to reverse back to a normal structural state once in a degenerative state.[10,83,84]

However, the emergence of an additional tendinopathic stage on the continuum, 'reactive-on-degenerative tendinopathy' has been supported by evidence demonstrating portions of degenerative tendon that are surrounded by structurally intact tendon.[10] The hypothesis around the mechanism of 'reactive-on-degenerative tendinopathy' is that structurally intact tendon assumes the bulk of loading (as degenerative tendon is mechanically dormant) via stress-shielding, and therefore may pass through intermittent stages of reactivity. Recent work by Docking et al (2015) demonstrates that degenerative tendon compensates for its inert structure by increasing cross-sectional area to maintain adequate aligned fibrillar structure around the periphery of the degenerative region.[80] As a result, the tendon maintains its capacity for load-transmission and can be targeted through load-based therapies. Importantly, sound progressive loading paradigms that address both pain and load-capacity in reactive-on-degenerative tendinopathies should be prescribed in order to maximize tendon resilience and prevent progressive structural pathology and functional disability (Figure 2.1).

In summary, while tendon structural abnormalities on imaging do not always correlate with the presence of tendon pain and dysfunction,[82,85] the progression and worsening of tendon structural pathology over time has been shown to be the greatest risk factor for the development

of pain.[86,87] Therefore, clearly defining the symptom and structural characteristics that may influence the progression of tendinopathy is of critical importance in the management of tendinopathy.

Figure 2.1. The continuum of tendon pathology (image from: Cook et al, *Br J Sports Med,* 2016)[10]



Differential Diagnosis: A Critical Feature of Evaluation of Anterior Knee Pain

One of the key components to identifying appropriate treatment pathways for individuals with patellar tendinopathy is a sound differential diagnosis. In the context of the proposed study, clearly defining diagnostic criteria for patellar tendinopathy is a critical feature of pursuing the study aims to compare landing biomechanics between distinct pathologic and healthy populations.

Common Differential Diagnoses for Non-Traumatic Anterior Knee Pain

Patellar tendinopathy is one of several conditions that presents clinically as non-traumatic anterior knee pain. The breadth of differential diagnoses is expected, due to the multiple anatomic tissues located around the knee joint and the biomechanical demands placed on the knee joint during activity. The most common differential diagnoses for non-traumatic anterior knee pain include, but are not limited to: patellofemoral pain syndrome, medial plica syndrome,

chondromalacia, pes anserine or supra/infrapatellar bursitis, iliotibial band syndrome, fat pad impingement, and osteoarthritis.[88,89] Furthermore, in pediatric and adolescent populations, additional differential diagnoses must be considered, including osteochondroses (Osgood-Schlatters disease and Sinding-Larsen-Johansen disease, osteochondritis dissecans, inflammatory disorders, referred pain (i.e. slipped capital femoral epiphysis or Perthe's disease), osteosarcomas, or patellofemoral instability.[88,89] In both clinical and research environments, a thorough clinical exam to rule-out conditions that are non-mechanical in nature and require referral should always occur first. If patellar tendinopathy is suspected, a systematic assessment of signs and symptoms should be confirmed.

Defining Tendon Pathology: Evidence from Previous Literature

Symptom Characteristics Associated with Patellar Tendinopathy

Patellar tendinopathy is characterized by several hallmark features that have been well-described in previous literature[11,67,90]: 1) localized pain at the proximal patellar tendon, just inferior to the inferior patellar pole[11,91], and 2) load-dependent pain that is provoked with high demands on the knee extensor mechanism.[5,92,93] Specifically, load-related tendon pain typically occurs immediately upon the initiation of loading, and resolves once the load is removed or at rest. Tendon pain also increases with increasing load magnitude and loading rates of activities involving high energy storage and release across the extensor mechanism, such as during deep squatting or repetitive hopping.[11,94] Occasionally, during the course of an exercise bout, tendon pain may subside, as the tendon "warms-up"; however, pain typically reemerges following cessation of activity and may last for several hours to days.[94]

Both pain location and dose-dependent nature of pain are critical features that differentiate tendinopathy from other common anterior knee pain conditions, such as

patellofemoral pain syndrome (PFPS) or infrapatellar fat pad (IFP) impingement. Unlike patellar tendinopathy, PFPS is characterized by diffuse anterior knee pain under or around the patella, provocation with lower-loading activities and prolonged knee flexion positioning, and reduction of symptoms with joint alignment correction, such as via taping or manual repositioning.[11,70,89,95,96] Additionally, while there is evidence of tissue communication between the IFP and the patellar tendon[97] (i.e. cytokine production,[98] neovascularization,[76,99,100] and structural connections[75,101102]), pain derived from IFP impingement is typically located adjacent to the tendon and is more commonly provoked with knee hyperextension or direct palpation.

Finally, in the context of evaluating young athletes with suspected patellar tendinopathy, developmental conditions involving tendon-growth plate interfaces, such as Osgood-Schlatters disease (tibial tuberosity) and Sinding-Larsen-Johansson disease (inferior patellar pole), should be considered. These two developmental, traction-apophysitis conditions are most common in pre-pubertal cohorts during periods rapid growth.[89,103] In the present study, the pubertal status of invited participants will be confirmed using validated measures[104–106], with the goal of minimizing the potential that either of the aforementioned developmental conditions may be the source of patellar tendon pain in the symptomatic group.

The criteria selected to characterize patellar tendon pain and delineate a symptomatic group assignment for participants in the current study are supported by previous literature (Table 5). Tendon pain is most commonly utilized clinically to diagnose patellar tendinopathy, regardless of the degree of structural pathology.[107] While the pathophysiological theories for the pain associated with patellar tendinopathy are complex and multi-factorial,[82] identification of key clinical features that can be easily quantified by clinicians to classify tendon pain is essential. Traditionally, positive pain on palpation was considered the hallmark sign of patellar

tendinopathy. However, the sensitivity (68%) and specificity (9%) of palpation is poor, and thus palpation is not considered as a robust diagnostic tool.[108] In addition to patient history, including detailed questioning of activity-related stimulants of symptoms, pain maps[109] and provocation tests[110] are commonly used to differentiate patellar tendon pain from other conditions.

Table 2.2. Criteria to characterize symptomatic patellar tendinopathy in current study

Criteria for Symptomatic Patellar Tendinopathy	Key Previous Literature	Notes
1. **Localized Load-Dependent Pain**	Blazina et al. (1973) Roels et al. (1978)	Stages 1-4 (pain location/provocation)
	Cook et al. (2000)	Junior basketball athletes
	Rudavsky & Cook (2014)	Topical review
2. **Single-limb decline squat (SLDS) pain that remains localized to tendon**	Purdam et al. (2003)	Adolescent (14-18 years) male and female basketball athletes
	Malliaras et al. (2006)	Adult volleyball athletes
	Cook et al. (2005)	Adult volleyball athletes

Blazina et al (1973) outlined the first diagnostic classification system for tendinopathy, highlighted by subjective report of symptoms during jumping and landing sport activities.[91] Due to the non-specific nature of global measures of this subjective assessment, additional studies sought to develop more objective measures, including standard squatting. Though a common functional test utilized in clinical evaluations, a standard squat is considered inadequately specific as a patellar tendon pain provocation test due to a lack of sufficient knee extensor mechanism loading. During a standard squat, end-range active ankle dorsiflexion motion is typically achieved prior to sufficient knee extensor mechanism loading.[110] However, Purdham et al (2003) demonstrated that a single-limb decline squat (SLDS) (Figure 2.2) had superior ability to discriminate a change in pain scores following heavy training in adolescent athletes, compared to a standard double limb or single limb squat on a flat surface.[110] Furthermore, the authors

emphasize that when using the SLDS provocation test, the test should be conducted with participants squatting between 50-60 degrees of knee joint flexion,[110] where maximum force development through the patellar tendon has been reported.[111] Additionally, larger magnitudes of knee flexion excursion (70-80 degrees) engage the patellofemoral joint and result in peak patellofemoral compressive forces[112]; therefore, avoiding a knee flexion angle greater than 60 degrees during the SLDS test should be confirmed. The SLDS has been reported to have a low standard error of 5% on repeated assessment.[110]

Figure 2.2. The Single Leg Decline Squat (SLDS). Participants are instructed to squat to approximately 60 degrees of knee joint flexion on a 25-degree decline board and rate the magnitude and location of pain. (image from: Malliaras et al, *J Orth Spor Phys Ther,* 2015)[11]

In summary, clear definitions for inclusion criteria to define symptomatic patellar tendinopathy are critical to determine the independent influence of pain and structural pathology on primary biomechanical outcomes of interest, and, furthermore, the effectiveness of the isometric patellar tendon loading exercise protocol.

Structural Characteristics Associated with Patellar Tendinopathy

Structural pathology is the second hallmark sign utilized to define tendon pathology. Ultrasonographic (US) and magnetic resonance imaging (MRI) techniques are used clinically for diagnosis, monitoring treatment outcomes, and to predict future symptom development.[113,114] It should be stated that, as with the utilization of diagnostic imaging for any medical condition,

imaging should always be accompanied by a sound history, clinical exam, and evaluative testing. The goal of diagnostic musculoskeletal imaging is to visualize bodily tissues in order to objectively characterize features that may be indicative of pathology.

Overview of Tendon Composition

Healthy tendon is primarily comprised of Type I collagen which is highly organized and well-aligned in parallel longitudinally within the tendon, providing tendon with high tensile strength.[115] Small amounts of Type III collagen and Type X collagen are also present in tendon, primarily at tendon-bone interfaces.[116] Tendon fibroblasts, or *tenocytes*, are spindle-shaped cells located along collagen fibers, and are the primary cell modulating the tendon structural environment via mechanotransduction.[12,117] Proteoglycans (primarily decorin and biglycan) support the tendon extracellular matrix, aid in regulation of collagen fiber formation, and control fibril diameter.[118] Proteoglycans and water constitute a large majority of the tendon extracellular matrix. In healthy tendon, the biochemical contrast between collagen and water in tendon results in little to no signal on MRI, and a homogenous, parallel orientation of well-organized fibrillar structure on US.[119]

Compositional Changes Associated with the Development of Tendinopathy

Several key histologic features of structural tendon pathology are commonly described in the literature that can be quantified using diagnostic imaging modalities. Driven by increased number of cells and alterations in cell shape (rounded tenocytes), these changes include: tendon thickening, changes in echogenicity, fibrillar disorganization, and neovascularization.[119] On imaging, pathological tendon typically appears with localized widening of the tendon, focal hypoechoic regions, irregular fiber structure, and/or neovascularization.[120] While MRI and US imaging modalities are adept at identifying neovascularization in tendon, for the purpose of this

study, the presence or absence of neovascularization will not be utilized as an inclusion criteria for a patellar tendon abnormality. Detection of neovascularization via Doppler signal on US can demonstrate poor inter-day reliability,[121] and as it is easily influenced by exercise.[122] Additionally, the selection of US for use in the current study is attributed to its accessibility, feasibility, and ease of interpretation. While MRI is reported to have excellent reproducibility, soft tissue contrast, and captures multi-planar images,[113] it is costly, inaccessible, and not required for the purposes of the study aims. Furthermore, advances in ultrasonographic imaging, including probe technology and image processing software that allows thorough visualization of tendon fibrillar structure, suggest that US imaging is an efficacious tool in tendon imaging.[114]

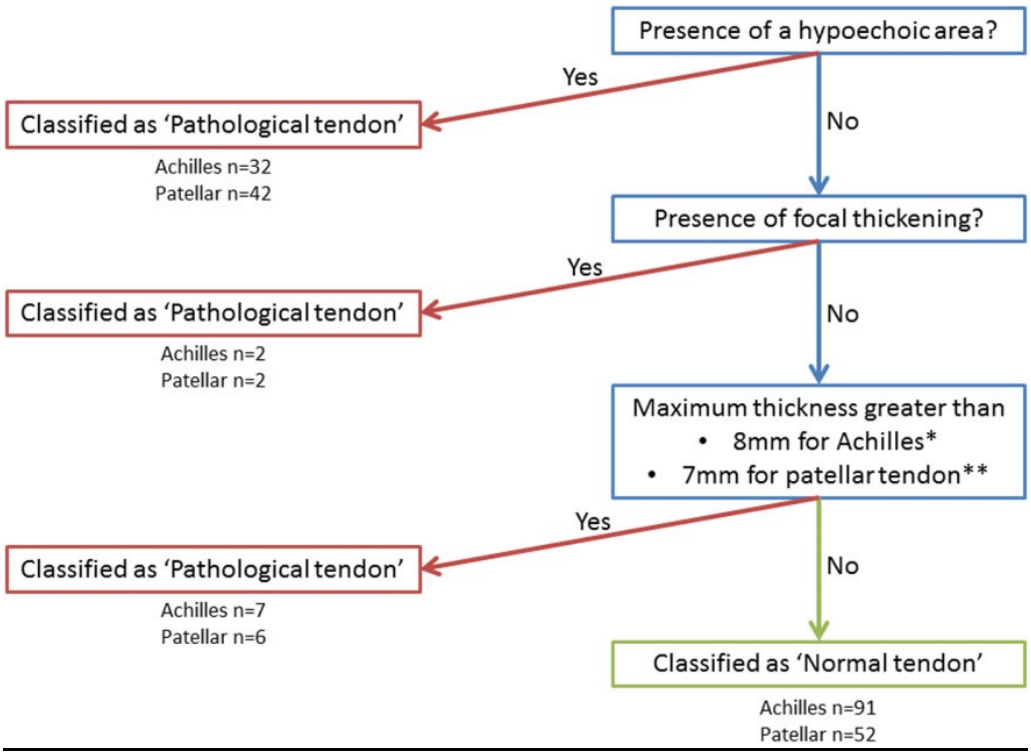
Ultrasound Assessment of Tendon Structure

Historically, conventional ultrasound imaging techniques have been utilized to evaluate tendon status, through measurement and grading of cross-sectional area (CSA) and echogenicity.[119,123,124] Hypoechoic regions are suggestive of poor tendon quality, and have been linked to symptomatology in numerous populations.[125,126] Cook *et al*.[1] demonstrated that in junior basketball players, 79% of the patellar tendons categorized on clinical evaluation as having "current tendinopathy" also had a hypoechoic region on US imaging. Additional ultrasonographic assessments, including Doppler sonography, have been utilized to investigate neovascularization of tendon, and have been shown to associated with the presence of symptomatic Achilles tendinopathy.[3,121,127]

However, the inconsistent presence of structural pathology and clinical symptoms of tendinopathy is well-accepted in current literature and clinical practice.[1,41,128,129] Khan *et al.*[130] found only moderate associations between conventional US findings and clinical assessment of individuals with chronic Achilles tendinopathy, and that baseline US findings did not predict

two-year patient-reported function. Additionally, in a group of competitive club runners, while conventional US detected increased tendon thickness, there were no associations with self-reported symptoms of Achilles tendinopathy.[129] Limitations to conventional US include error related to probe placement and handling during scan acquisition, and slight changes or error in transducer position (tilt and rotation) can generate anisotropy artifact that may mimic images visualized with actual pathology.[114] However, a recent systematic review found that diagnostic US assessments for tendon size demonstrate acceptable inter- and intra-rater reliability.[131] Therefore, based on the evidence, the use of US to classify tendon pathology (Figure 2.3) in the current study is well-supported.

Figure 2.3. Algorithm for criteria decision-making to characterize patellar tendon structural pathology using conventional ultrasound imaging in current study (from Docking and Cook, 2015).[80]



Application to Subject Inclusion Criteria: The Importance of Clear Definitions for Patellar Tendon Pain and Structural Pathology

Utilizing a systematic approach to the evaluation of tendon pain and tendon structural pathology is an important feature of the current study. Previous research demonstrates varied outcomes when assessing biomechanical characteristics of individuals with tendon pain and/or structural pathology. For example, Rosen et al. (2015), having defined load-dependent pain as pain with jumping or squatting activities in the previous three months, found lesser peak knee and hip flexion angle and knee and hip flexion displacement during a landing task than asymptomatic controls.[21] However, Siegmund et al (2008), having defined tendon pain only on point-tenderness on palpation, reporter greater hip flexion angle and acceleration during a jumping task than asymptomatic controls.[20] These divergent findings provide an example of how observed outcomes may differ when consistent, clearly defined criteria for symptomology are not upheld. Therefore, the aim of the current study is to utilize evidence-based, systematic criteria to define participant status and group assignment (symptomatic with PTA, asymptomatic with PTA, healthy control) in order to elucidate the independent effects of pain and structural pathology on key biomechanical outcomes. Support from previous literature for the key biomechanical outcomes of interest for the current study will be outlined in the following section of this review.

SECTION 2: Intrinsic and Extrinsic Factors Associated with Patellar Tendinopathy

Numerous studies have investigated factors, including intrinsic and extrinsic factors, associated with the development of patellar tendon structural pathology and symptomatic tendinopathy. Intrinsic risk factors are factors or characteristics inherent to the individual, such as anthropometrics and biomechanics. Extrinsic risk factors are factors or characteristics external

to the individual but which may influence or act on the individual, such as training load. When interpreting risk factors, it is important to consider the association between a factor with tendon structural pathology and/or symptomatic tendinopathy, as these two outcomes do not always occur concurrently.

Intrinsic Factors

Multiple studies have investigated whether certain anthropometric factors increase or decrease an individual's risk of patellar tendinopathy.[92] Interestingly, the association between anthropometric characteristics and patellar tendinopathy is not consistent. Lesser quadriceps[132] and hamstring[2,132] extensibility have been associated with structural pathology and a higher risk for symptomatic patellar tendinopathy. Waist girth (>83cm) has been identified as a risk factor tendon structural pathology in males, which is thought to be driven by both mechanical and biochemical effects of heavier weight.[133] Other anthropometric characteristics, such as lesser closed-chain ankle dorsiflexion range of motion,[134] lower arch height,[135] younger age, and taller height[4] have all been associated with patellar tendinopathy. Vertical jump height is a common performance measure assessed in athletes, and several studies have demonstrates associations between greater jump height performance and patellar tendinopathy[36,136] and structural pathology.[2]

An individual's biomechanical profile is another type of intrinsic factor associated with patella tendinopathy. Biomechanics are believed to be particularly important given in regards to tendinopathy as an individual's biomechanics directly influences tendon loading and is highly modifiable. Biomechanical profiles that characterize risk factors and injury mechanism factors associated with lower extremity musculoskeletal injury are readily reported in the literature. Existing biomechanical literature is especially robust in the area of biomechanical factors

associated with traumatic knee injuries, specifically anterior cruciate ligament (ACL) injury. The use of biomechanical analyses allows for the quantification of multiple aspects of human movement, including kinematics and kinetics, to better understand injury risk factors and mechanisms, and to inform prevention strategies for both primary and secondary injury events. An important distinction to acknowledge when assessing and conducting biomechanical analyses is that between risk factor analysis and mechanism analysis. *Injury risk factor analysis* utilizes prospective study design, first identifying biomechanical characteristics of an individual's movement and then monitoring that individual over time to record any subsequent injury events.[137] From this design, risk factors can be identified and used to help predict future injury. *Mechanism analyses* seek to evaluate biomechanical variables that are present at the time of an injury event.[138] In other words, the mechanistic approach quantifies how excessive stress to a biological tissue leads to tissue failure and injury. Clear expression of biomechanical profiles as either injury risk factors or injury mechanism factors is critical. In his model of sports injury prevention, van Mechelen (1992) outlines the necessity of clearly identifying and delineating risk factors and mechanisms that influence injury in order to develop appropriate targeted injury prevention strategies.[139] In the context of this review, risk factors and mechanistic factors associated with patellar tendinopathy will be clearly defined.

Compared to the large volume of biomechanical literature around ACL injury, there is much less available concerning biomechanical factors associated with patellar tendinopathy. Additionally, biomechanical literature around patellar tendinopathy typically only evaluates individuals with structural pathology or tendon pain compared to asymptomatic healthy controls, which limits direct comparison of the independent and combined influence of structural pathology and pain on biomechanics. Furthermore, diagnostic criteria for structural pathology

and pain frequently differ between studies, making comparison and integration of reported outcomes difficult. Finally, the majority of studies utilize small sample sizes and very specific participant populations (i.e. elite sport-specific athletes), which decreases external validity of findings. Nonetheless, it is clear from current literature that there are not clear landing strategies between individuals with patellar tendinopathy and in those that go on to develop patellar tendinopathy. Due to the load-responsive nature of tendon and high propensity for developing tendinopathy with high, repetitive external loading, understanding how landing characteristics differ between individuals at different stages of the tendinopathic continuum is important to inform injury prevention and best practice rehabilitative strategies.

An important consideration when using biomechanical profiles to assess severity of a MSK injury is that biomechanical characteristics of a given task do not always correlate with performance of the task. This has been demonstrated in numerous studies examining individuals with patellar tendinopathy, where no differences in vertical jump height performance exists between groups, despite different biomechanical strategies.[220] For example, the symptomatic basketball athletes studied by Siegmund et al. compensated with altering ankle and hip kinematics to achieve similar jump-heights as the asymptomatic athletes. That said, jump height may be a critical factor in predisposing an athlete to future tendinopathy due to the high knee extensor moments and subsequent patellar tendon loading. In a 5-year prospective study of elite adolescent volleyball,[140] male athletes who went on to develop symptomatic tendinopathy performed significantly better on a baseline countermovement jump (38.0±5.8 cm) than those who remained asymptomatic (34.6±5.5 cm; p=0.03). Furthermore, after accounting for gender and years of volleyball training, the odds of developing symptoms were 2.09 (1.03-4.25) for every one centimeter of additional height on the countermovement jump.

The following portion of this review aims to summarize the current body around biomechanics in individuals with symptomatic and/or structural patellar tendinopathy in order to inform the key biomechanical variables of interest selected for the current study.

Ankle, Knee, and Hip Kinematic & Kinetic Characteristics Associated with Symptomatic Patellar Tendinopathy

The following section will describe lower extremity kinematic and kinetic characteristics that have been associated with patellar tendinopathy. Kinematic characteristics of highly dynamic movements, such as jumping and landing, are relevant in the context of patellar tendinopathy based on the anatomy of knee extensor mechanism. Designed as a "pulley system" across the anterior knee, the patellar tendon experiences high tensile loads as the knee joint moves through increasing degrees of sagittal plane motion. However, at certain regions of the patellar tendon, specifically as the tendon wraps around a bony segment (i.e. proximal patellar tendon at the inferior patellar pole), high compressive loads are added to the overall load in the tendon. Combined tensile and compressive load has been described as a key mechanical factor leading to tendinopathy,[141] as evidenced by alterations in tissue composition from fibrous to fibrocartilaginous, changes in tenocyte shape, and an increase in aggregan and type II collagen in areas of compression within tendon.[142,143] Additionally, knee joint angle directly influences the line of action of the patellar tendon relative to the longitudinal axis of the tibia, such that contraction of the knee extensor mechanism will either invoke anterior (more extended knee) or posterior (more flexed knee) movement of the tibia.[144] Furthermore, the patellar tendon is oriented anteriorly at knee flexion angles less than 60° and posteriorly as knee joint flexion increases,[145] which may lead to greater compressive stress on the posterior aspect of the patellar tendon by the patella. The variable change in patellar tendon moment arm distance throughout

different degrees of sagittal plane joint motion directly influences the amount of quadriceps muscle force production required to combat external knee flexion moment on the lower extremity from the ground reaction force.[144,146,147] Therefore, it is hypothesized that kinematic patterns, particularly sagittal plane motion at the knee, are associated with the development of patellar tendinopathy, and that once symptomatic, alterations in joint motion may exist as a compensatory movement strategy due to pain.

Kinematics Associated with Symptomatic Patellar Tendinopathy

Biomechanical assessments of kinematic variables are most commonly reported from landing assessments via comparisons of individuals with and without symptomatic patellar tendinopathy. Sagittal plane knee joint motion is the most commonly cited variable, as the majority of energy absorption and force generation at the knee occurs in the sagittal plane,[148] and do the aforementioned implications of sagittal plane knee position on patellar tendon strain. In several studies, individuals with symptomatic patellar tendinopathy demonstrate trends towards lesser sagittal plane ankle, knee, and hip motion than asymptomatic healthy control participants.[19,20] However, in a their systematic review and meta-analysis, van de Worp et al. report the inconsistencies in landing kinematics between those with and without symptoms. [93] It is possible that slightly differing definitions for symptomology between the study's cohorts may contribute lack of large group differences in these previous studies, further warranting the importance of clear and consistent definitions of pain when conducting studies on pathologic populations.

In a more recent study, utilizing the largest comparative cohort size in current patellar tendinopathy biomechanics research (30 recreationally-active participants per group), Rosen et al (2015) found that symptomatic participants, compared to asymptomatic participants, landed with

lesser peak knee joint flexion (74.8±13.2° vs. 82.5±9.0°, respectively) and had lesser sagittal plane knee joint excursion (71.6±8.4° vs. 79.7±8.3°, respectively) during the stance phase of a jump-landing task.[21] Additionally, the symptomatic participants demonstrated lesser peak hip flexion and hip flexion excursion than the asymptomatic participants. Interestingly, no differences knee and hip frontal and transverse plane kinematics, nor in tri-planar ankle kinematics were noted between groups, suggesting that adaptations in sagittal plane motion in the presence of tendon pain may be the dominant movement compensation strategy chosen by those with symptoms to reduce combined tensile and compressive tendon loads. Sorenson et al (2010) found no statistically significant differences in sagittal plane knee joint excursion between symptomatic and asymptomatic elite male volleyball athletes.[19] However, the lack of group differences in this study may be attributable to lack of statistical power (n=7 and n=6 athletes per group, respectively), the inclusion of one athlete with bilateral pain, and the nature of the task (single limb approach jump analysis).

Similarly, symptomatic versus asymptomatic elite dancers performing a "saut-de-chat" single-limb landing technique demonstrated similar sagittal plane ankle, knee, and hip joint angles at initial contact ($p>0.05$), varying by less than two degrees at each joint between groups. In this study, the landing approach angle was calculated as the angle between the floor and the L5-S1 reflective marker at the time of initial contact to generate an estimate of gross body position during the landing task.[149] Interestingly, despite no statistically significant group differences in landing angle, landing angle explained 67% of the braking impulse value (r=-0.817), with symptomatic participants demonstrating greater braking impulse even at similar landing angles as asymptomatic participants. It is possible that these individuals chose a movement pattern to avoid greater sagittal plane knee joint excursion to minimize symptom

exacerbation and compensated with higher braking forces across the lower extremity. This finding provides further support of the relationship between compensatory kinematic and kinetic patterns in athletes with patellar tendon pain.

Knee joint velocities and accelerations during landing have also been previously explored in this population. In a matched comparative cohort of adult male basketball athletes, those with symptomatic jumper's knee demonstrated decreased knee flexion acceleration and a longer-duration landing time than healthy controls, despite no group differences in knee joint flexion velocity or displacement during a countermovement jump.[20] Additionally, the symptomatic athletes demonstrated greater maximum hip and trunk flexion angle and hip flexion acceleration than healthy controls, which is suggestive of a strategy to decrease the moment arm between the trunk and knee.[20] This positioning subsequently reduces the external knee flexion moment imposed on the lower extremity at ground contact, thus reduced the necessary internal knee extension moment and extensor mechanism force production requirement.[150] Similarly, Bisseling et a. (2007) found that symptomatic volleyball athletes landed with lower knee velocities and slower ankle plantarflexion and knee extension moment development.[22] Applying force over a longer period of time reduces the magnitude of an applied load (F=dt), and may therefore reduce the stress applied to a given tissue, in this case the painful patellar tendon.[146] Evaluating joint accelerations may have implications in further identifying compensatory movement strategies employed by individuals with patellar tendon pain.

Associations between ankle joint dynamics and knee loading is a commonly investigated biomechanical concept with regards to lower extremity injury.[148,151–154] Existing literature around patellar tendinopathy consistently demonstrates that ankle kinematics do not differ between symptomatic versus asymptomatic individuals.[21,155] Rosen et al (2015) found no significant

differences at the ankle in any plane-of-motion at initial contact, peak, or maximum angular displacement in recreational athletes performing a double-limb jump-landing task.[21] Similarly, in a study by Richards et al (2002), albeit a very small sample size of asymptomatic (n=7) and symptomatic (n=3) national elite male volleyball players, no differences in ankle kinematics were noted between groups during a spike jump task.[155] Interestingly, in a blinded logistic regression model, the only ankle kinematic or kinetic variable to correctly predict the presence or absence of patellar tendinopathy in this cohort was foot inversion moment. In a previous study of the same cohort of athletes investigating the predictive capacity of knee dynamics for current patellar tendinopathy, Richards et al (1996) found that a combination of kinematic and kinetic variables correctly predicted the presence or absence of tendinopathy at the time of testing, including larger knee flexion angle at initial contact, peak vGRF and vGRF loading rate, internal knee extension moment development, and tibial external rotation moment.[156] The collective results of these studies suggest that there is likely a combination of ankle and knee dynamics that are linked to patellar tendinopathy. However, due to the cross-sectional nature of these study, the ability of these biomechanical factors to predict which athletes will go on development tendinopathy in the future is not feasible.

Kinetics Associated with Symptomatic Patellar Tendinopathy

In addition to the few number of studies that have reported differences in sagittal plane knee kinematics between individuals with and without tendon pain, kinetic variables have also been explored in this population. It is important to recognize analyses being conducted on double- versus single-limb tasks, as outcomes may differ based on the nature of the task. Both Bisseling et al. (2007) and Sorenson et al. (2010) analyzed double-limb landing tasks, and demonstrated that symptomatic athletes tend to choose a load-avoidance strategy, supporting the

expected notion that individuals avoid loading painful tissues during high-energy movements. Bisseling et al.[22] demonstrated that symptomatic volleyball athletes chose a landing technique that avoids high patellar tendon loads, specifically with lesser internal knee extension moment on the involved limb and reduced knee flexion velocity, compared to both those with a history of patellar tendinopathy and healthy control participants. Though their study utilized a small sample size (N=13), Sorenson et al.[19] found that individuals with patellar tendinopathy had approximately a 30% reduction in net joint work and power and approximately 22% lower peak vGRF than healthy control participants, suggesting reduced mechanical energy absorption and force attenuation on the symptomatic limb.

Conversely, during the single-limb saut-de-chat in dancers, dancers with symptomatic patellar tendinopathy demonstrated greater peak vGRF (36%) and vGRF impulse (15%) and greater posterior GRF (82%) and posterior GRF impulse (126%) compared to healthy controls.[149] It is possible that during single-limb tasks, when an individual does not have another limb to rely upon, distinguishing movement characteristics that may precede or be associated with the presence of pain is more overtly recognizable. In this study, higher lower extremity loading during a single-limb task in the symptomatic group may suggest that individuals with patellar tendon pain are less capable of attenuating external forces when required by the nature of the task to rely solely on the symptomatic limb. However, as with all non-prospective studies of individuals with pain or pathology, a direct cause and effect relationship cannot be established; therefore, we cannot conclude whether or not symptom onset drives biomechanical alterations, or conversely, if aberrant biomechanics lead to the development of pain and structural pathology.

Ankle, Knee, and Hip Kinematic & Kinetic Characteristics Associated with Patellar Tendon Structural Pathology

Due to the increased likelihood of developing symptomatic patellar tendinopathy when a PTA is present,[43,157] evaluating the biomechanical profiles of an asymptomatic with PTA group provides an opportunity to gain insight into movement characteristics that may precede symptom onset. However, there are very few previous studies have investigated kinematic and kinetic characteristics of individuals with patellar tendon structural pathology.

In a recent systematic review by van de Worp et al. (2014), a quantitative analysis determined that greatest differences in kinematic and kinetic variables during landing tasks were present in studies that compared healthy controls to asymptomatic individuals with patellar tendon structural abnormality (PTA).[93] Previous work has compared biomechanics during both horizontal and vertical landing phases of jump-landing tasks. The majority of previously reported differences between individuals with a PTA and controls are during *horizontal* landing phases, in which individuals are moving forward. These differences are thought to be attributed to greater magnitudes of posteriorly directed ground reaction forces during horizontal landings, requiring greater internal knee extension moment to decelerate the limb and prepare for subsequent vertical movement.[25,97]

Furthermore, Edwards et al. (2012) demonstrated that peak patellar force is higher during the horizontal deceleration phase versus vertical phase of a landing task.[158] Therefore, it is necessary to consider the type of landing task being analyzed in existing literature, and the presence or absence of group differences may be influenced by task specificity and mechanical demand. Additionally, current evidence indicates that future research examining landing mechanics that may be associated with the development or progression of patellar tendinopathy

should include a dynamic task that incorporates a horizontal landing component, as the demand of the horizontal task on the MSK system may better differentiate factors associated with tendinopathy.

In a comparative cohort of junior pre-elite male basketball athletes with and without PTA, several kinematic variables were found to be predictive of the presence of a PTA, including hip joint excursion and knee flexion angle at initial ground contact. Individuals with a PTA extended their hip joints when landing and had greater knee flexion angles at initial ground contact.[24] A similar hip kinematic pattern has been noted in adult athletes with PTA performing the same task.[23] Combined with greater hip flexion angle at initial ground contact,[23,24] this movement strategy is thought to increase the demand on the knee extensor mechanism to counteract the more posterior position of the body's center of mass when transitioning from the loading to propulsive phases of a jump. Edwards et al. (2010) also found that the adult male athletes with PTA demonstrated greater knee flexion angles at initial ground contact but moved through less knee flexion excursion throughout the loading phase. Interestingly, neither of these studies reported internal knee moments, which have been shown to differ between individuals with and without patellar tendon pain.[19,22] Further investigation of differences in internal knee and hip joint moments, moment development, and energetics between individuals with and without PTA is warranted, as high internal loads to the patellar tendon are thought to be associated with the progression of structural pathology and pain.

Patellar Tendon Biomechanical Loading Characteristics Associated with Patellar Tendinopathy

The knee extensor mechanism must deal with high magnitudes of kinetic energy during landing tasks, with upwards of 7x BW of force placed on the patellar tendon.[23,159] The high and

frequent application of patellar tendon load during sport-specific activities has been associated with the development of tendinopathy.[156] Recent literature has suggested that methods that directly estimate patellar tendon load are more specific than using traditional kinetic assessments, such as ground reaction forces, and may better discriminate the magnitudes of tendon load in one type of landing task compared to another and between pathological groups.

The characterization of patellar tendon loading during landing tasks has been investigated in several previous studies. In a group of asymptomatic male athletes with normal patellar tendon structure (confirmed via ultrasound), Edwards et al. (2012) quantified traditional kinematics and kinetics, as well as estimated patellar tendon loads, during the horizontal and vertical phases of a stop-jump task.[158] Several findings from this study provide important insight regarding relevant variables of knee joint loading when studying chronic overuse injuries such as patellar tendinopathy.

First, in a comparison of the two landing phases, individuals demonstrated greater peak patellar tendon force (F_{PT}), patellar tendon force loading rate (LR F_{PT}), peak posterior ground reaction force, and peak internal knee extension moment during the horizontal landing compared to the vertical landing. Interestingly, these outcomes were observed despite lower peak vGRF during the horizontal landing phase.[158] These findings suggest that in biomechanical analyses, using the peak vGRF as a surrogate for F_{PT} may largely underestimate the actual load placed on the patellar tendon, further justifying the methodology of estimating patellar tendon force.[144,160] Additionally, this study justifies the inclusion of loading rate variables, not just of ground reaction forces but also of the force development within the patellar tendon, as both the magnitude and rate of force development has implications to create tissue overloading.

Furthermore, Janssen et al. (2013) investigated whether factors previously associated with the development of tendinopathy, including demographics, clinical measures, strength, and biomechanics, affect patellar tendon loading during dynamic tasks. In this group of elite, healthy volleyball athletes performing a lateral stop-jump task, male participants with greater quadriceps strength, increased ankle dorsiflexion velocity, and increased trunk flexion velocity were predicted to sustain higher patellar tendon forces (R^2 = 52.0%) and faster patellar tendon force loading rate (R^2 = 69.8%).[26] This study provides further evidence of the utility in quantifying patellar tendon force variables in this population.

Understanding the function of the patellar tendon during physiological loading conditions is important when studying knee biomechanics. This knowledge may be especially relevant in the study of patellar tendinopathy pathogenesis and treatment, as the nature of applied load to tendon tissue is associated with tendon's adaptive properties, particularly when exposed to highly repetitive loads.[83]

Advances in dynamic imaging technology has permitted three-dimensional modeling of human subjects in vivo during tasks reflective of every-day movements. DeFrate et al. (2007) quantified three-dimensional patellar tendon kinematics during a weight-bearing single-limb lunge in healthy participants (no knee pathologies).[145] There are several key findings from this study that are of relevance to this review. First, as the knee moves through flexion (0-110°) the patellar tendon was found to change its orientation in both the sagittal and coronal plane. Large changes in patellar tendon length were observed from 0-30°, and subsequently very small changes in length from 30° to 110°, supporting previous literature that has reported greater patellar tendon stiffness at increasing knee flexion angles. Though modelling the patellar tendon two-dimensionally, Edwards et al. (2012) found that peak F_{PT} for both the horizontal and vertical

landings occurred between approximately 55-60°.[158] These studies provide support for the propensity for patellar tendon overloading during dynamic tasks involving repetitive sagittal plane knee joint motion. Furthermore, pairing knowledge of tendon kinematics and kinetic under physiological loading conditions with three-dimensional lower extremity joint kinematics and kinetics may provide a more comprehensive approach to understanding movement strategies of individuals at risk for or with patellar tendinopathy.

To this end, Edwards et al (2010) investigated landing strategies of asymptomatic athletes with a confirmed PTA compared to healthy controls, including both a traditional kinematic and kinetic assessment as well as estimation of patellar tendon force.[23] Interestingly, during both horizontal and vertical stop-jump tasks, the PTA and healthy control groups demonstrated no statistically significant differences in peak FPT, LR F_{PT}, or peak vGRF. However, the PTA group employed a different overall landing strategy at both the knee and hip during the horizontal landing phase. Specifically, the PTA group had greater knee flexion at initial ground contact but lesser knee flexion excursion, greater hip flexion at initial contact but lesser hip flexion excursion, and earlier recruitment of the hamstring muscles compared to the controls.[23] The authors suggest that this altered kinematic strategy in the PTA group may result in higher compressive and tensile loads on the patellar tendon due to the distribution of the center of mass and subsequent combined loads and demand on the knee extensor mechanism. From a muscle recruitment standpoint, early hamstring recruitment at larger hip and knee flexion angles at initial contact is thought to invoke a large posterior shearing forces across the knee joint, thereby increasing the demand on the knee extensors to counteract this knee flexion moment. This study provides important insight into a compensatory landing strategy adopted by individuals with patellar tendon structural pathology who are not yet symptomatic. Furthermore, it suggests that

there is value in developing strategies to identify compensatory movement patterns in high-risk individuals such that injury prevention strategies can be optimally implemented.

Application to Selection of Biomechanical Variables of Interest

What remains to be investigated is how kinematics and kinetics, including patellar tendon force, may differ between individuals with a confirmed PTA but with or without symptoms compared to healthy controls. The biomechanical variables selected for evaluation and analysis in the current study (Tables 1.1 and 1.2) will allow thorough examination of three key characteristics related to the knee extensor mechanism function and patellar tendon pathoetiology, including:

1) Sagittal and frontal plane knee motion, which influence the line of action and moment arm of the patellar tendon force vector, as well as the imposed combined tensile and compressive forces on the tendon
2) Kinetic variables (internal moments and ground reaction forces), which describe the loading demands (force-generation requirements) on the knee extensor mechanism.
3) Patellar tendon force variables, which have been demonstrated to provide a more specific estimation of the actual load placed on the tendon during dynamic tasks, therefore serving as an important adjunct variable of interest to traditional kinetic variables (internal moments and ground reaction forces).

Exploring landing strategies of these specific groups will improve the ability of clinicians to develop targeted, individualized interventions for implementation based on where an individual is on the continuum of tendon pathology.

Extrinsic Factors

Extrinsic factors, such as training surface and shoe wear have not been identified as risk factors, despite their clinical relevance as risk factors for other MSK overuse conditions, such as lower extremity bony stress injuries.[161,162] Perhaps the most consistently identified extrinsic factors associated with tendinopathy is an individual's training load. Specifically, an individual's training magnitude, frequency, and duration have been associated with tendinopathy in multiple studies.[34,61,69,70] In elite adolescent volleyball athletes, high training load volume (hours and sets per week) (OR: 1.72-3.38) and years of volleyball participation (OR: 2.22) associated with an increased risk of patellar tendinopathy.[36] Furthermore, Visnes et al (2013) found that in elite adolescent volleyball players followed prospectively for five years, those that developed symptomatic patellar tendinopathy (n=28) trained significantly more (14.4±2.5 hours/week) compared to those who remained asymptomatic (n=122) (11.8±2.7 hours/week; p=0.001).[140] In adult female basketball athletes, those with structural pathology trained approximately 2.6 more hours per week and reported lower self-reported physical function than those without pathology.[163] Further evidence in the Achilles tendon literature demonstrates significantly higher cumulative incidence of both Achilles tendinopathy (adjusted OR: 31.2) and tendon rupture (adjusted OR: 14.9) before the age of 45 in former male elite long distance runners and sprinters, respectively, compared to non-athletes.[164] Training load, therefore, is identified clearly in existing literature as a critical factor associated with the pathoetiology of patellar tendinopathy.

Physical Activity and Training Load Monitoring in Sport

The concept of monitoring athlete training load in sport has been of interest to performance specialists for many decades, specifically from the perspective of maximizing human physical performance capacity and recovery.[165] The earliest concepts of training load

monitoring in sport is best illustrated endurance sports, such as distance running, in which methodology was developed to manipulate and quantify the components of training that we now refer to as the FITT principle (frequency, intensity, time, type).[165] From these early efforts came advanced metrics by which training loads can be assessed more thoroughly and objectively, prompting an exciting area of research and practice for clinicians and scientists.[165]

The categories and definitions commonly used in load monitoring literature must first be defined to ensure consistent interpretation of outcomes. *External load* is defined as any external stimulus applied to the individual measured independently of internal characteristics, such as duration of game (minutes) distance run (miles), or steps-per-day. *Internal load* is defined as the relative physiological and psychological response experienced by an athlete in response to an external load, such as heart rate, rating of perceived exertion (RPE), blood lactate concentrations.[165–168] Numerous benefits of athlete load monitoring have been established, including explaining changes in performance, understanding training responses, and identifying areas of potential non-functional overreaching.[168] The desire to measure and monitor both external and internal training load is of interest to coaches, athletes, medical practitioners, and researchers alike who have a collective interest in performance, injury prevention, and recovery. While load monitoring may not exclusively explain an athletes' response to exercise, the quantification of objective and subjective characteristics of an individuals' load response provides greater insight into factors that may be associated with the cascade of overreaching / overtraining, or the development / progression of a musculoskeletal injury.[167,169]

There are emergent recommendations for load monitoring to become a regular component of managing all patients, not just elite athletes; this particularly applies to patient populations where the mismanagement of load may have deleterious effects on the trajectory of

recovery and long-term resilience.[170] Recent research has demonstrated strong associations between external training loads and injury incidence in elite adult athlete populations,[34,35,165,171–173] specifically explored in cricket, soccer, and Australian football. The interplay between training load and injury development / progression characterizes the accepted models for the development of overuse injuries, such as patellar tendinopathy, as its pathoetiology thought to arise from load mismanagement.[9,12,68,117] However, emerging evidence reiterates that high training volume and intensity are not always deleterious; specifically, moderate training loads may be protective against injury to certain tissues, which has been described by Tim Gabbett as the "training injury prevention paradox".[171,173] Both high and low external loads have been associated with injury risk,[174] suggesting that there may be an optimal load threshold for individual athletes, consistent with Dye's Envelope of Function[64] and the inverse-*U* illustration. This recent work in training load management has led some to believe that "overuse" may not be the best terminology to characterize non-traumatic injury development; instead, the terminology "training load error" injury may be more appropriate.[175]

Physical Activity and Training Load Monitoring of Individuals with Lower Extremity Musculoskeletal Injury Conditions

The use of wearable technology to quantify objective load metrics, both in clinical and research settings, has grown exponentially in the last two decades.[176] The majority of studies to-date have been designed to better quantify the influence of physical activity on systemic health outcomes, such as obesity, cardiovascular health, and diabetes, in both younger and older populations.[177–180] Interestingly, the application of wearable technology in the context of musculoskeletal injury has been slower to develop, but its momentum has increased due to advances in technology.[167] Implementation of wearable technology in an athletic environment

can be challenging due to constraints that may include time, manpower, lack of knowledge, or financial constraints;[167] however, recent work supports the utility and relevance of external load monitoring-based data to the understanding of factors that may be related to injury incidence, as well as outcomes following injury.

The majority of the existing training load literature has explored the aforementioned relationships in adult populations. In general, there is a lack of evidenced-based support for whether these same relationships exist in a younger adolescent population. Several studies report training load variables in specific populations of young athletes, including youth track and field athletes,[181] baseball pitchers,[182] and cricket fast bowlers,[183] have demonstrated associations between high training loads or insufficient recovery and injury. As described in this review, the development of patellar tendinopathy in athletes is likely as result of a "training load error", and its progression related to the mismanagement of external load over time. In an effort to better understand the factors related to the development of patellar tendinopathy, as well as how individuals with varying levels of the condition (structural +/- symptoms) may differ, objectively quantifying and monitoring external load is critical. The youth population may be especially vulnerable to training load errors as they transition from junior to senior level play, participate on multiple teams (i.e. school and club based) simultaneously, and mature physically following puberty.

A study conducted by Visnes and Bahr (2013) tracked training volume prospectively for four-years via self-reported training diaries in elite volleyball players aged 16-18.[36] The development of symptomatic patellar tendinopathy associated with higher overall training volumes, specifically number of hours and matches played. This important study supports the relevance of quantifying load variables in a young population at risk for patellar tendinopathy. A

limitation of this study is that the diagnosis of patellar tendinopathy was made via self-reported history of pain for twelve weeks and tenderness to palpation, with no quantification of pain on loading (i.e. single-limb decline squat) or assessment of tendon structural abnormalities. Therefore, the question still remains regarding how training load may associate differently in athletes across the continuum of tendon pathology (e.g. pain and/or structural abnormalities) and, furthermore, if potential differences in cumulative external load associate with differences in biomechanical movement profiles that are modifiable through structured interventions.

Esmaeili et al. (2016) only found small and inconsistent effects of training load on Achilles and patellar tendon structure.[184] However, the measure of training load in this study was session rating of perceived exertion (sRPE); while a valid and reliable measurement that associates with injury risk and fatigue in athletes,[167,185] sRPE may not be directly related to changes in tendon structure as it does not directly quantify a specific measurement of cumulative external load, such as steps-per-day. Therefore, quantify cumulative external load in a population of individuals with tendinopathy is still needed.

The use of load monitoring to describe populations with MSK injury conditions is an exciting area of research and is likely to add to the body of literature seeking to understand factors related to long-term health outcomes. As described previously in this review, the ability to quantify variables related to "real-world movement" is a critical missing piece that may be imperative to better understand the trajectory of recovery, resolution of impairments, and durability patients over time, particularly once they are no longer involved in clinical-supervised rehabilitation. Additionally, monitoring load in a real-world environment provides unique insight into an individual's activity in the setting in which they spend the majority of their time versus

exclusively evaluating and developing management strategies based on laboratory or clinical evaluations alone.

In a recent study, Bell et al. (2017)[38] took this exact approach to explore differences in physical activity between individuals following ACL reconstruction (ACLR) and healthy matched controls. This study utilized the ActiGraph wGT3X-BT accelerometer over a one-week monitoring period, which is the same methodology proposed in the current study. Both steps-per-day and minutes of moderate-to-vigorous physical activity (MVPA) were significantly lower in the ACLR group compared to the health control group, despite groups reporting similar levels of physical activity on self-reported function questionnaires.

The findings from this study have several important implications in the context of the current study. First, Bell et al (2017) demonstrated the feasibility of utilizing a hip-worn ActiGraph accelerometer in a young, active population for a one-week monitoring period. Additionally, it appears that cumulative external load and self-reported function do not always associate, further supporting the importance of not relying exclusively on patient self-reported function in the context of understanding the influence of the patient condition / pathology on real-world physical activity. This may be particularly important in a youth population, as recall of prior physical activity may be unreliable,[165] and due to the varied nature of a young individual's daily life (i.e. attending classes, extra-curricular activities, sports activities, etc.). Finally, the design of and findings from this study are in line with the aims of the current study. Though performed in a post-ACLR population, it is the first to objectively quantify cumulative external load in a pathologic population in which long-term outcomes have been readily associated with tissue (in this case, knee joint) response to load over time. The current study aims to utilize a similar approach to determine if there are differences in cumulative external load

between individuals with symptomatic PTA, asymptomatic PTA, and asymptomatic without PTA for the purpose of better understanding modifiable factor, particularly that manipulation of cumulative loading, that may move an individual along the continuum of tendon pathology.

Application to Physical Activity Monitoring Methods

The application of physical activity monitoring is relatively new in the context of musculoskeletal injury and rehabilitation, yet its relevance to patellar tendinopathy is clear. Strong evidence discussed in the prior section supports the association between high training loads and a greater risk for the onset or progression of patellar tendinopathy in young athletes. Therefore, using objective variables, such as steps-per-day, to further quantify cumulative loading past traditionally-measured variables, such as years of participation and duration of practice/competition, may help better describe associations between physical activity and tendinopathy. Finally, using a hip-mounted accelerometer used to assess physical activity over a one-week monitoring period is accepted in the current literature as an appropriate wear position and duration, respectively, to obtain an estimate of cumulative physical activity in a youth population. The current study is the first to our knowledge to apply physical activity monitoring methods to a young population of individuals with patellar tendinopathy. With the increased availability and societal use of physical activity monitors, there is an exciting opportunity for clinicians to utilize this technology to improve both prevention and treatment strategies for musculoskeletal conditions, moving movement analysis from strictly laboratory-based assessments to clinical and real-world monitoring strategies.

SECTION 3: Exercise-Based Intervention Paradigms for Patellar Tendinopathy

Mechanotherapy: Implications for Treatment of Tendinopathies

As introduced in Section 1, biological tissue homeostasis has been described by Dr. Scott Dye (1996) as the *envelope of function,* or "the range of load that can be applied across an individual tissue in a given period without supraphysiologic overload or structural failure."[64] This model outlines the relationship between magnitude and frequency of load to illustrate optimal tissue function when maintained within the zone of homeostasis. In the zone of homeostasis, there is appropriate external loading to foster internal responses that promote positive tissue adaptation. In this model, Dye identifies four key areas: 1) under-loading, 2) homeostasis, 3) supra-physiological load, and 4) structural failure (Figure 2.4).[64]

As with any living organisms, human musculoskeletal tissues are most efficient and best protected when operating under homeostatic conditions. When the stress to a tissue exceeds its internal tolerance, or ability to withstand a load, it enters a zone during which temporary or structural micro-failure or permanent injury may occur. Conversely, reducing the magnitude or frequency of load to the tissue can result in under-loading of the tissue, which, in the case of tendon, has been shown to result in a loss of mechanical strength and even structure if unloading continues for a prolonged period of time.[117]

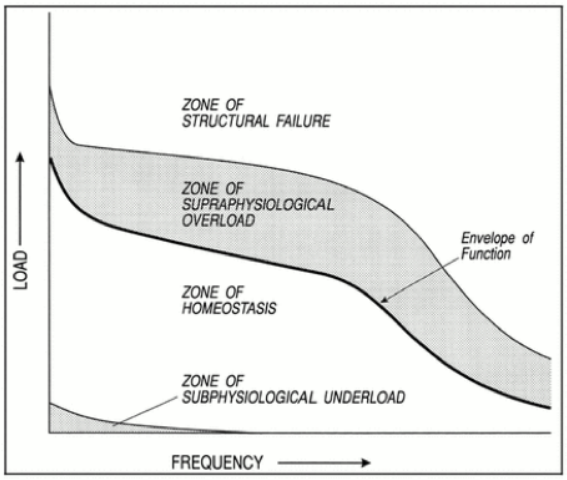


Figure 2.4: The Envelope of Function[64]

Overuse injuries occur when loading exceeds a tissue's homeostatic boundary. Similarly, a tissue may be under-loaded, such as in the case of lower extremity muscle atrophy due weight-

bearing restrictions. Individuals who develop symptomatic knee injuries may have exceeded the load tolerance across the joint, leading to tissue breakdown. The knee, which can be described as the transitory joint between the femur and tibia, is highly responsible for dissipating lower extremity loads; therefore, in the presence of aberrant biomechanics, the knee may be highly vulnerable to injury. Furthermore, following knee injury, it may be critical to reestablish tissue homeostasis in order to correct biomechanical faults and prevent progression of pathologic processes.

Tendon health is intimately related to mechanical homeostasis; therefore, Dye's envelope of function model is readily applicable to the development of tendinopathy. Tendon adaptation occurs through mechanotransduction, the physiological process by which the body translates mechanical load into a cellular response that leads to structural change.[12] Mechanotransduction is characterized by three key components[12]:

1. *Mechanocoupling*: the literal perturbation of a cell when a load is applied. This perturbation stimulates the release of calcium and other cytokines into the gap junctions to initiate the cascade of events across cell membranes.
2. *Cell-to-cell communication*: the cellular communication between cells. A message from the cell receiving the direct stimulation (location 1) travels via gap junctions and ion channels to the next cell (location 2), which is rapidly repeated. These mechanisms are responsible for how the "load message" is relayed from one area of the tendon to another.
3. *Effector response*: the response of cellular tissue receiving the message to create either an anabolic or catabolic response

Mechanotransduction is the underlying mechanism for how mechanical load influences a tissue's cellular response. *Mechanotherapy* is, therefore, a term used to describe how load can be

manipulated to influence the mechano-transductive events within a tissue. In his review of Mechanotherapy application in rehabilitation, Khan suggests that understanding the cellular response to loading should be at the foundation of how clinicians prescribe both rest and tendon-specific loading.[12]

Historical Perspective: Eccentric Exercise for the Treatment of Tendinopathies

Combining knowledge of the continuum of tendon pathology with an understanding of the cellular response of tendon to loading provides the rationale for exercise prescription for individuals with tendinopathy. Eccentric-based strength-training protocols have traditionally been utilized in the treatment of chronic tendinopathies,[13–15] particularly following the landmark study by Alfredson in the late 1990s, from which developed the "Alfredson eccentric exercise" programs used commonly in clinical practice and research investigations.[66]

However, recent research suggests that loading interventions, including type and dosage of exercise, should be prescribed based on where a tendon falls on the continuum of pathology. Cook et al. (2016) suggests that 'phenotyping' patients based on the presence of pain, structural abnormalities, and dysfunction may help clinicians direct patient-centered treatments and improve short- and long-term patient outcomes (Figure 2.1).[10]

For example, the prime instigator of a reactive tendinopathy is a rapid increase in loading without adequate recovery time, such that there is an acute inflammation in the paratendon (synovial sheath) and initial disruption of the tendon matrix.[9] Here, eccentric exercise, which involves large stresses and resulting strain to tendon tissue, is typically not tolerated and may perpetuate symptoms. Instead, a period of initial rest is appropriate in order to reduce or remove the offending load and allow adjacent inflammation to subside, followed by the gradual re-introduction of controlled loading.

Conversely, in the degenerative stage of chronic tendinopathy, eccentric exercise has been shown to be effective at reducing pain,[13–15,186] increase force, stiffness, and Young's elastic modulus,[187] decreasing tendon thickness,[79] and improved self-reported function.[79] It is thought that the high tensile stress placed on the tendon during eccentric loading helps to stimulate the tendon extracellular matrix (which has reparative capability) and maximize the performance of residual healthy tissue. Studies tracking tendon structural response to cumulative load demonstrate the capacity for aligned fibrillar structure to compensate for areas of disorganization and degeneration by increasing cross-sectional area to maintain sufficient volume of load-responsive tissue.[10,80] From a treatment perspective, it is thus recommended that load-based interventions for tendons in this stage should target building load-capacity in the aligned portion of the matrix, or "treating the doughnut, not the hole".[10,80] By selecting the appropriate type of load-based intervention for individuals with patellar tendinopathy, it is possible to reduce pain and/or improve tendon load capacity, which may aid in improving the effectiveness of other traditional interventions, such as neuromuscular re-education.[82,188,189]

Therefore, it is evident that load-based exercise interventions should be selected based on patient characteristics such that implementation is successful. In a youth athlete population, this is particularly important, as achieving compliance with rehabilitation interventions can often be difficult. In the context of the current study, it is essential to select an intervention that will be tolerated by individuals with tendon pain and / or structural abnormalities.

Treating Symptomatic Patellar Tendinopathy: Evidence for Isometric Loading Exercise

Emerging evidence supports the use of isometric loading exercise for individuals with symptomatic patellar tendinopathy. The impetus to explore isometrics as a treatment option stemmed from the fact that eccentric loading protocols are often painful to complete and

therefore met with poor compliance when implemented in-season for individuals with patellar tendinopathy.[14,90] Since the onset of symptomatic tendinopathy in athletes is not necessarily a season-ending injury, the goal of treatment is to minimize pain in order to facilitate continued sport participation.

The rationale behind using isometric exercise can be summarized in two categories: mechanics and neuromuscular characteristics. During isometric exercise, particularly when contractions occur over a prolonged time-period, time under tension is maximized, leading to greater tendon strain. As a viscoelastic tissue, longer duration and heavier, isometric contractions generate larger strain in the tendon tissue, which is thought to be the stimulus for tendon adaptation.[190–192] Additionally, in mid-ranges of knee joint flexion motion, the length-tension relationship of the quadriceps is maximized, such that optimal muscle force can be generated. The percentage of motor unit activation has been shown to be greater during isometric versus concentric and eccentric contractions.[193] Furthermore, compressive stresses on the patellar tendon are less at mid-ranges of knee flexion compared to the extremes of knee joint flexion.[144,145] Therefore, mechanically, loading the tendon isometrically in a mid-range of knee flexion has mechanical advantages. In a population of young individuals, who may have a greater potential for tendon adaptation, the ability to respond positively to an isometric loading protocol may be even more likely.[194]

Secondly, isometric exercise is thought to be effective due to its ability to improve neuromuscular function via modulation of central-mediated inhibition. Previous studies in healthy participants have demonstrated that isometric muscle contractions reduce pain pressure thresholds.[195,196] In the context of patellar tendon pain, it is theorized that the presence of pain, both at rest and during / following exercise, may alter cortical representation, which may alter

motor output via changes in the excitatory and inhibitory neural pathways.[17] While it is well-accepted that exercise can change voluntary activation in various,[197–201] this is a relative new area of investigation in tendinopathy. However, pivotal work by Rio and colleagues over the last five years has provided support for the effectiveness of isometric exercise in patellar tendinopathy patients. In a within-respondents, single-blinded randomized cross-over trial studying male volleyball athletes with symptomatic patellar tendinopathy, Rio et al (2015) investigated both the effectiveness of and mechanisms of cortical motor function following both isometric and isotonic loading exercise protocols.[17] While both protocols were well-tolerated, a one-time acute bout of isometric loading exercise demonstrated superior outcomes compared to isotonic exercise in improved pain relief, improved self-reported function, increased quadriceps strength, and decrease in cortical inhibition.[17] Furthermore, pain reduction and quadriceps strength gains were sustained 45 minutes following the isometric protocol but not the isotonic protocol. The same isometric loading protocol has also demonstrated positive outcomes in pain reduction and excellent patient compliance and tolerance when implemented in-season with athletes with symptomatic patellar tendinopathy.[16]

Application to Isometric Loading Intervention

In summary, recent evidence demonstrates the effectiveness of isometric loading to improve quadriceps strength and decrease pain in individuals with symptomatic patellar tendinopathy. Additionally, this isometric loading protocol is tolerated by patients with symptoms who are still participating in sport when implemented in-season. However, the effects of this targeted tissue-specific loading protocol on lower extremity biomechanics has not yet been investigated. Athletes with symptomatic patellar tendinopathy demonstrate load avoidance movement strategies during sport-specific tasks, including reductions in sagittal plane knee

displacement and mechanical energy absorption, lesser vertical ground reaction force, and lesser internal knee extension moment.[19,21,22] This study will be the first to test the acute effects of an isometric patellar tendon loading exercise protocol[17] on landing biomechanics in individuals across the tendon pathology continuum (both asymptomatic and symptomatic individuals with structural abnormalities). Using isometric loading interventions to acutely change movement biomechanics may provide an important next step in rehabilitation paradigms for tendinopathy as a method to promote improve load-tolerance, and stimulate positive mechano-transductive responses in individuals with tendon pathology.

CHAPTER 3: EXPERIMENTAL DESIGN & METHODS

SUBJECTS

A total of 45 male participants from the high school, club, and collegiate sport population in the Raleigh-Durham-Chapel Hill area and the University of North Carolina at Chapel Hill were recruited for this study. Male athletes participating in sports involving large volumes of jumping and landing maneuvers were eligible for participation, as this cohort has been identified to develop patellar tendinopathy most readily.[5,67] Specifically, eligible participants wer of a post-pubertal age through 28 years of age. Post pubertal age was defined based on the Pubertal Development Scale (PDS), a valid and reliable questionnaire-based, non-invasive assessment utilized in previous literature to assess pubertal status.[105,106,202] (Appendix 1) Furthermore, participants were actively participating within an organized sport setting (high school, collegiate, club, competitive intramurals) in their respective team's weekly training and competitions, quantified by a Tegner Activity Level Scale of ≥ 5 (Appendix 2). Additionally, eligible participants demonstrated either the presence or absence of patellar tendon symptoms (SYM vs. ASYM) and patellar tendon structural abnormality (PTA) for group assignment (described in *Session 1: Screening Session* to follow).

The following exclusion criteria were applied to all participants:

1. Any anterior knee pain with no patellar tendon abnormality on ultrasound assessment.
2. History of any knee joint surgery ever.
3. History of other (non-knee) lower extremity surgery in the last 1 yea

4. History of lower extremity injury in last 6 months (other than patellar tendinopathy).
5. An injection (corticosteroids, plasma-rich-protein, etc.) to the patellar tendon in the last 3 months.
6. Known history of osteoarthritis or current symptoms related to osteoarthritis (i.e. stiffness, swelling).
7. Participation in formal rehabilitation for patellar tendinopathy in prior 3 months.
8. Known neurological disorders, including: stroke, multiple sclerosis, ALS, diabetic neuropathy, epilepsy, traumatic brain injury resulting in loss of consciousness, concussion within the last 6 months, cranial neural surgery, balance disorders.
9. Use of pacemaker or another implantable electronic device.
10. History of cardiac arrhythmia or any cardiac condition.
11. History of psychiatric disorder.
12. History of cancer in the brain or thigh musculature.
13. Pubertal Development Score <12 (Stages 1-4).

Participants were recruited via email correspondence and informational packet/flyer distribution to local high school, club, collegiate, and competitive intramural teams by the principal investigator (PI) (L.S.P.). The PI also made in-person recruitment announcements with teams during team meetings and training sessions or following competitions. During recruitment and initial screening (in-person, over phone, email), the PI ensured that participants were in-season prior to being invited to the laboratory for Session 1.

DATA COLLECTION

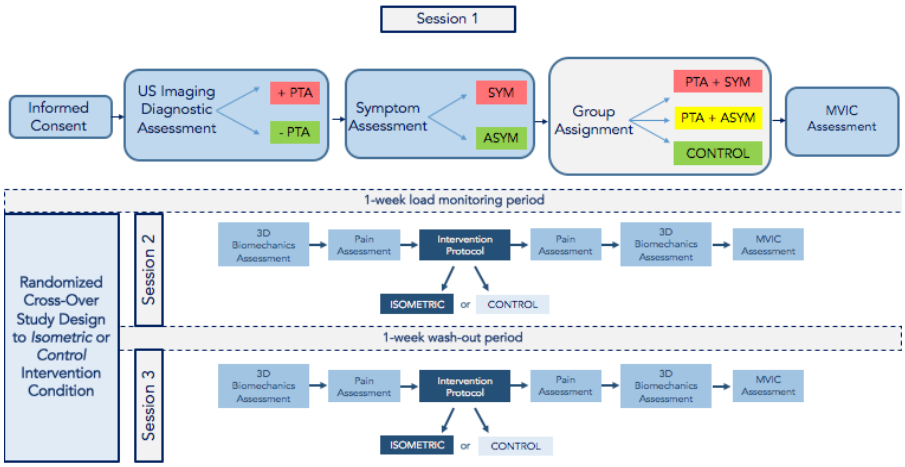
Procedures

The study procedures involved three visits to the Sports Medicine Research Laboratory (SMRL) for testing (Sessions 1-3). Session 1 was termed the Screening Session, while Sessions 2-3 were termed the Intervention Sessions. Each session was separated by 7-10 days.

Overall Study Design

We utilized a cross-sectional design to assess baseline movement profiles (Aim 1) and one-week cumulative physical activity load volume using wearable technology (Aim 2), and a randomized cross-over study design to assess acute intervention effects on lower extremity kinetic and kinematic biomechanical variables during landing (Aim 3), A diagram outlining the overall study design is provided in Figure 3.1. A detailed explanation of each session is described in the next portion of this chapter.

Figure 3.1. Overall study design diagram.



Session 1: Screening Session

The following procedures outline the screening session. This session took approximately 30-45 minutes to complete.

Participant Demographics, Inclusion / Exclusion Criteria Screening

Upon the interested study candidate's arrival to the Sports Medicine Research Laboratory, the PI verbally confirmed that the candidate met the appropriate demographic inclusion / exclusion criteria. Included in this screening material was the Pubertal Development Scale (Appendix 1) to ensure that both age and pubertal status were quantified prior to study enrollment. Any candidates who scored at a Stage 1-4 (PDS score <12) were considered to be pre-pubertal or pubertal and were excluded from the study. Any candidates who scored at a Stage 5 (PDS score >12) were considered to be post-pubertal and therefore met the maturity level criteria for the study. If a candidate met all of the aforementioned study inclusion / exclusion criteria, he was invited to participate in the study and completed Institutional Review Board documentation, physical activity questionnaire (Appendix 3), injury history form (Appendix 4), percentage of predicted mature height[104] (Appendix 5) and the Victorian Institute of Sport Assessment-Patellar Tendon (VISA-P) questionnaires (Appendix 6).[203,204] Additionally, participant height and weight was obtained with shoes removed, and information regarding leg dominance (the self-reported limb the participant would choose to kick a ball for distance) was recorded.

Ultrasound Imaging Screening

A LOGIQe US system (General Electric Co., Fairfield, CT) with a high-resolution 12 MHz linear probe was utilized to image the patellar tendon. A single investigator (L.S.P.) performed the examination to image right and left patellar tendons of each participant. The participant was positioned on the treatment table in supine with the knee flexed to 90° in order to place enough passive tension on the extensor mechanism and avoid possible anisotropy of the patellar tendon when concave in full knee extension.[205] The location of the inferior patellar pole

was marked manually with a pen based on palpation and confirmed during scanning. Ultrasound gel was applied to the participant's anterior knee to improve contact between the probe and skin. The US scanning parameters were set to: frequency=12MHz, depth=3cm, gain=50.

First, the US probe was placed longitudinally in line with the mid-portion of the proximal patellar tendon just inferior to the inferior pole of the patella. The probe was tilted medially / laterally such that it was perpendicular to the long axis of the patellar tendon to maximize image quality. Once the appropriate angulation / tilt of the probe was achieved, the image was frozen and saved for analysis. While the probe was in this longitudinal position, the inferior patellar pole and a standard distance of 1 centimeter distal to inferior patellar pole were marked on the skin, respectively. This location was utilized to acquire transverse ultrasound images. Next, the US probe was centered transversely at the inferior patellar pole and tilted superiorly / inferiorly such that it was parallel to the short axis of the patellar tendon, and once appropriately positioned, the image was frozen and saved for analysis. The same procedure was followed at a standard distance of 1 centimeter distal to the inferior patellar pole, respectively. Three longitudinal and three transverse images of the patellar tendon were acquired. These procedures were repeated for the contralateral limb, such that bilateral patellar tendon US images were collected.

Maximum length, width, and height (cm) of hypoechoic regions were measured in both the sagittal (longitudinal) and axial (transverse) planes using ImageJ software (National Institutes of Health, Bethesda, MD). All US images were taken by the principal investigator (L.S.P.), who is trained in ultrasonographic assessment of tendon. An expert co-investigator (D.B.) was consulted for any questionable images to confirm the presence or absence of PTA based on the

aforementioned criteria. These procedures have been reported in previously published literature.[80,85,205]

Symptom Assessment

Following the US assessment, participants were assessed for the presence or absence of patellar tendon pain bilaterally through two methods:

1. The participant was asked if he have any knee pain. If he answer "yes", the principal asked the participant to point to the location of the pain and the location will be recorded by the principal investigator on a knee image on the data collection sheet. If he answer "no", this answer was recorded.
2. The participant was asked to complete a single-limb decline squat (SLDS)[110] on a 25° degree decline board (Figure 2.2) to approximately 60° of knee flexion. The participant was first asked to rate any pain present on a standard numeric rating scale (NRS: 0-10), where 0 = no pain, and 10 = worst possible pain.[17,206] The participant was presented with a pain-map diagram immediately following the conclusion of the SLDS (Appendix 6).[110] On this diagram, the participant was asked to identify the location of pain during the SLDS. The participant chose from a series of pictures that were presented simultaneously in a grid-formation; the participant was able to view all pictures simultaneously in order to avoid any bias from the order in which the pictures were presented to the participant. The participant performed the SLDS bilaterally, and completed the same assessments for each limb. If the participant reported bilateral pain during the SLDS, the following operational definitions were utilized to determine the "involved limb" for the purposes of the study to ensure that the individual has a unilateral tendinopathy

a. The "worse" limb must be ≥ 5/10 on VAS Scale (0-10)

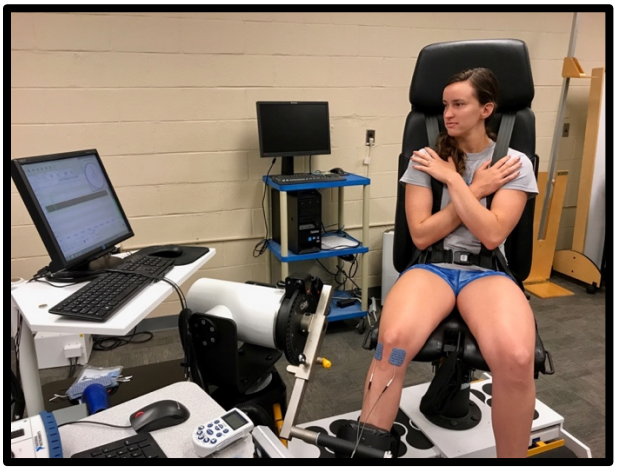
b. The contralateral limb must be ≤ 2/10 on VAS Scale (0-10)

Quadriceps Maximum Voluntary Isometric Contraction Assessment

Quadriceps strength was assessed by measuring the individual's maximum voluntary isometric contraction (MVIC). We proposed to collect the baseline MVIC during Session 1 instead of Session 2 in order to avoid any confounding effect of performing the baseline MVIC just prior to the intervention period, as it may influence the interpretation of the isolated effect of the isometric intervention protocol on the primary biomechanical outcome measures.

MVIC was assessed on the HUMAC Norm Dynamometer (CSmi, Stoughton, MA). This test was performed on the involved limb of participants with SYM-PTA and ASYM-PTA. All participants were positioned on the dynamometer with the test limb flexed to 60°. The knee flexion position was chosen in order to be consistent with previous studies investigating the effects of isometric loading in a patellar tendinopathy cohort,[17,18] and to minimize added compressive forces across the anterior knee at greater knee flexion angles (i.e. 90°) that may instigate pain during contraction.[141,145] The thighs, hips and torso were firmly stabilized with straps, and the arms will be folded across the torso to isolate the contribution of the quadriceps muscle without extremity movement (Figure 3.2). The lever arm was adjusted so that the ankle strap was placed 2 finger widths (~3 centimeters) proximal to the lateral malleolus. The knee was positioned so that the lateral femoral epicondyle was aligned with the rotational axis of the dynamometer. Measurements of chair position (distance of seat-back position, ankle strap position on lever arm) were recorded to ensure consistency between all subsequent assessments using the HUMAC system.

Figure 3.2. HUMAC Norm Dynamometer participant set-up for intervention



A series of graded submaximal warm-up isometric contractions were performed at 25%, 50%, and 75%, respectively, of the participant's perceived maximal effort but not recorded. The goal of this procedure was for muscle warm-up and participant familiarization with the task. For the MVIC testing, participants were instructed to "kick as hard and as fast as they can" into the dynamometer and to maintain maximal effort for approximately two seconds. The PI provided standardized verbal encouragement for each trial, and the participant received real-time visual feedback of their torque production on a computer monitor display directly in front of the dynamometer. Participants performed practice MVIC trials until the torque measurement fails to increase more than 10% from the previous trial. At this time, three maximum effort testing trials were collected. Each trial lasted approximately two seconds, and participants had sixty-seconds rest between each trial. If for a given trial, the participant was able to produce torque greater than 10% of the previous trial, this trial was repeated. The peak torque (N*m) of the MVIC from the three trials was recorded and averaged for analysis. Peak torque was expressed relative to body mass ($N*m*kg^{-1}$).

Post-Screening Session: Group Assignment

Following the conclusion of the screening session, participants were assigned to a group (SYM-PTA, ASYM-PTA, or CON). Participants that had symptoms and a patellar tendon abnormality were included in the symptomatic group (SYM-PTA). Participants that did not have symptoms but had a patellar tendon abnormality were included in the asymptomatic group (ASYM-PTA). Participants with no symptoms and no patellar tendon abnormality were included in the healthy control group (CON).

Group assignments were based on the following specific inclusion criteria:

Inclusion Criteria for Symptomatic Group with Patellar Tendon Abnormality PTA (SYM-PTA)

The following criteria must be met for symptoms (SYM):

1. Localized pain: Self-reported pain-location (single-finger pointing) at inferior pole patella or along the patellar tendon from inferior pole to tibial tuberosity
2. Single-limb decline squat (SLDS) pain that stays localized to inferior pole ("G" on pain map) or along the patellar tendon from inferior pole to tibial tuberosity ("E" on pain map). If the SLDS was positive for pain unilaterally, the painful limb was considered the "involved" limb and the non-painful limb was considered the "uninvolved" limb for the duration of the study. If the SLDS was positive for pain bilaterally, the following criteria was used to determine the "involved" and "uninvolved" limbs for the purpose of this study:
    1. The "worse" limb must be ≥ 5/10 on VAS Scale (0-10)
    2. The contralateral limb must be ≤ 2/10 on VAS Scale (0-10)

The following criteria must be met for patellar tendon abnormality (PTA):

A patellar tendon abnormality (PTA) was determined from the ultrasound images. A PTA will be defined as: 1) presence of a hypoechoic region ≥ 2mm, evident in *both* the longitudinal

and transverse scans, and/or 2) presence of focal thickening/fusiform swelling with or without hypoechoic areas, and/or 3) maximum thickness > 7mm. An algorithm for this criteria-based decision-making to characterize PTA is reported in previous literature by members of the current study team (Cook et al., 2013; Docking & Cook, 2015).[80,85] This algorithm for classification of patellar tendon abnormality is also supported by additional published studies.[43,75,207] The patellar tendon was classified as normal if all of these features are absent, and abnormal if a single or multiple feature(s) are present (Appendix 8).

Inclusion Criteria for the Asymptomatic Group with Patellar Tendon Abnormality (ASYM-PTA)

The inclusion criteria for the ASYM-PTA group are:

1. Absence of both localized self-reported pain and single-leg decline squat pain (as described above).
2. A unilateral patellar tendon abnormality (PTA) on US imaging, using the same criteria as described above.

Inclusion Criteria for the Health Control Group (CON)

The inclusion criteria for the CON group are:

1. Absence of both localized self-reported pain and single-leg decline squat pain (as described above).
2. Absence of a patellar tendon abnormality (PTA) on US imaging, using the same criteria as described above.

Once a participant's enrollment into the study was confirmed, the participant was assigned a study identification number which was used throughout the remainder of the study. Within each participant folder, there was a sealed opaque envelope with no external markings[208] and with a piece of paper enclosed with either 'A' or 'B' written on it, where A = isometric

condition, and B = control condition. These letters correspond to the intervention protocol that was conducted at the Session 2. For example, if 'A' was in the participant's envelope, the participant underwent the isometric condition at Session 2 and the control condition at Session 3.

Physical Activity Monitoring Period

Cumulative external load volume variables were collected during a one-week physical activity monitoring period between Sessions 1 and 2. As described above, participants were queried during the screening session to ensure that the one-week monitoring period was representative of a normal week of physical activity / sports training and competition in order to avoid capturing uncharacteristic weeks of physical activity (i.e. family vacation, week off of training/competition, etc.).

Following the conclusion of the screening session data collection (enrollment, ultrasound imaging, symptom assessment, quadriceps strength testing), participants were provided with an ActiGraph GT9X Link accelerometer (ActiGraph Corporation, Pensacola, FL). The GT9X is a solid-state tri-axial accelerometer, magnometer, and gyroscope with sampling frequency capability ranging from 30-100Hz with Bluetooth® capabilities to capture, record, and store high-resolution human activity information.[209] The ActiGraph GT9X Link accelerometer was selected for use in this study because it has been shown to be a valid and reliable accelerometer to capture objective data of steps-per-day and minutes of moderate-to-vigorous physical activity (MVPA) in young, active cohorts.[38,210,211] In a recent study by Bell et al. (2017), the ActiGraph wGT3X-BT accelerometer (the older model in the GTX line) was utilized and detected lower steps-per-day and MVPA in individuals following anterior cruciate ligament reconstruction (ACLR) compared to healthy controls.[38] In this study, step count was strongly associated with MVPA in both the ACLR group (r=0.914) and control group (r=0.877) ($p < 0.05$). Therefore, we

believed that the selection of this the ActiGraph accelerometer, which is the most commonly used validated accelerometer in physical activity research,[212] for our young, physically active population with and without pathology was appropriate to achieve the aims of the current study.

During the one-week monitoring period, participants were asked to wear the accelerometer over the right anterior superior iliac spine, affixing it to their clothing using an ActiGraph belt-clip (Appendix 9), for a period of seven-days, starting with the day following the screening session. A seven-day monitoring period is an accepted duration commonly utilized in physical activity literature.[38,39,211,213] A multiple-day assessment has been shown to improve reliability and decrease variability of objective physical activity data.[214,215] For the purpose of this study, a valid monitoring period was considered as a minimum of four total days, including three weekdays and one weekend day, for at least 8 hours-per-day, which is consistent with previous studies.[38,212,216] Participants were instructed that they could remove the accelerometer for bathing and sleeping, but that it should be worn at all other times throughout the day. Participants were provided with an individual docking station that could be connected via USB to a standard wall outlet for charging. Participants were asked to charge the accelerometer each night to ensure consistent battery life throughout daily wear periods.

During this instructional session, a demonstration was provided by the PI so that the participant felt comfortable with donning / doffing and charging procedures. The GT9X Link accelerometer was placed in a standard LCD display mode, showing only date, time-of-day, and battery life. The choice to not display the rolling steps-per-day count was so that participants were not biased by their daily performance; feedback of daily performance may challenge participants to perform more physical activity than normal due to external motivation. Additionally, participants were asked to keep a daily physical activity log to record type of

activity, time of day, duration (minutes), perceived intensity (modified RPE: 0-10)[217] and intensity category (light, moderate, or vigorous), and any relevant comments. This log was utilized to descriptively characterize physical activity features in conjunction with the primary dependent variable for this study, steps-per-day. The log also served as a quality-control check to ensure that participants were physically active at the minimum criteria within an organized sport setting (high school, collegiate, club, competitive intramurals) as outlined in study inclusion criteria. A recent study by Phibbs et al. (2017) demonstrated that a self-reported daily training log questionnaire, administered 24-hours after exercise, showed high levels of agreement with the criterion measure of session-rating-of-perceived-exertion (sRPE) administered within 30-minutes after exercise in adolescent athletes.[218] Therefore, the inclusion of a training log during the one-week load monitoring period was advantageous to capture self-reported activity data from participants.

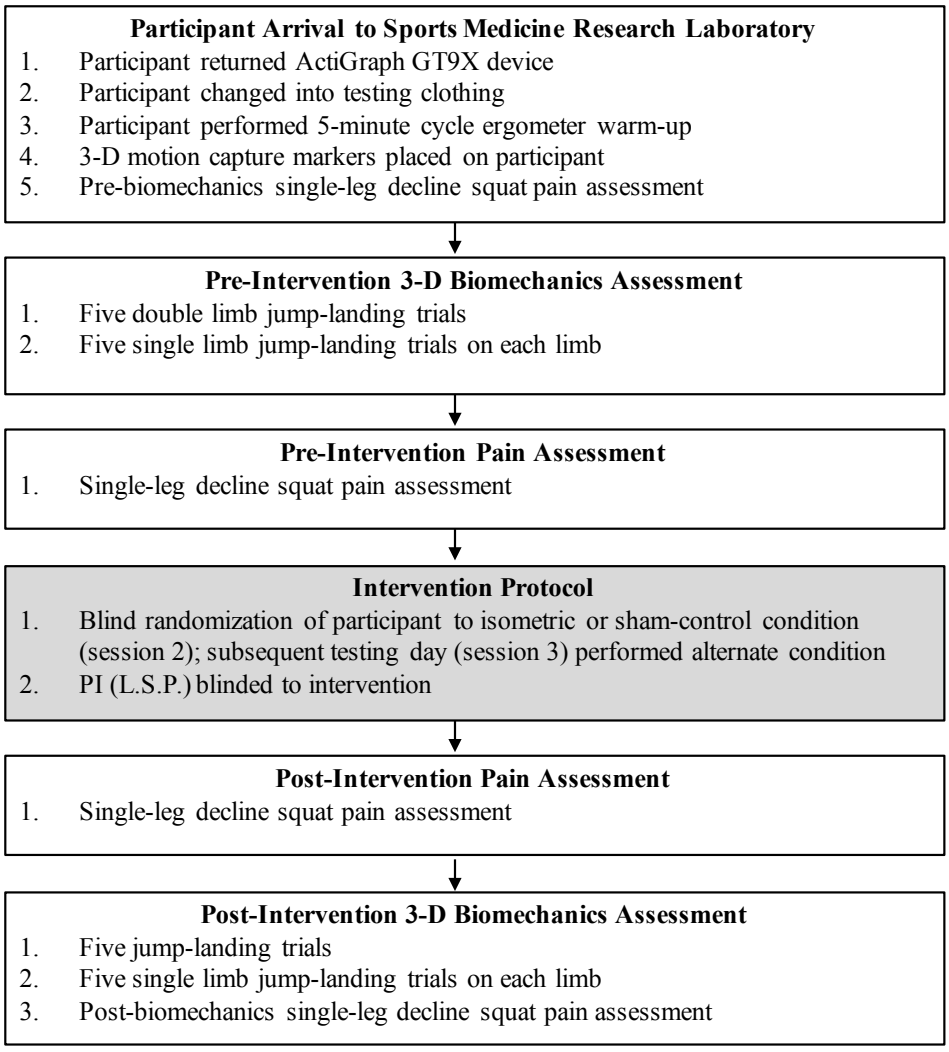
To maximize the acquisition of quality data, participants were provided with an instructional and troubleshooting guide and the PI's cellular phone number to utilize in the event of any problems that arose during the one-week monitoring period. The participant returned the accelerometer to the PI when he returned to the laboratory for Session 2. At this time, the data was evaluated for adherence to wear guidelines (days and hours-per-day). If wear guidelines were not met, the participant was asked to wear the accelerometer again for the one-week period between Sessions 2 and 3.

Sessions 2 & 3: Intervention Sessions

For Sessions 2 & 3, testing procedures and data collection flow were identical with the exception of the intervention protocol (isometric or sham-control). Session 2 & 3 each took approximately 1-hour to complete. A flow chart outlining data collection procedures for Sessions

2 & 2 is provided in Figure 3.3. Prior to the participant's arrival to the Sports Medicine Research Laboratory, a research assistant selected from a pre-filled envelope to determine the intervention condition assignment for Session 2 (see above). The principal investigator was blinded to this intervention order assignment.

Figure 3.3. Testing procedures for Sessions 2 & 3.



Upon arrival to the Sports Medicine Research Laboratory for Session 2, participants changed into black spandex shorts and tank-top. Participants wore personal athletic shoes during the entire testing protocol. After assuming the correct attire, participants completed a 5-minute warm-up on a cycle ergometer within the laboratory at a self-selected pace.

Three-Dimensional Biomechanical Assessment

Following the warm-up period, the participants were escorted to the motion capture area for the biomechanical assessment. Participants were then outfitted with 20 retro-reflective markers bilaterally on the following bony landmarks: acromion process, anterior superior iliac spine (ASIS), greater trochanter, medial and lateral femoral condyles, medial and lateral malleoli, calcanei, and the first and fifth metatarsal heads.[219] A single marker was placed on the manubrium of the sternum and at the L4-L5 vertebral space. Rigid clusters of three or four markers were placed at the sacrum and on the thigh, shank, and foot segments bilaterally. A static trial was captured with the participant standing with arms positioned at 90° of shoulder abduction to estimate the location of the landmarks needed to calculate joint centers.[219] After the static trial, the single markers on the foot, malleoli, femoral condyles, and greater trochanters were removed. If at any time during data collection, a marker(s) were to fall off of the participant, the marker(s) were replaced, calibration markers repositioned, and another static calibration trial captured. Attention was made to ensure that static calibration trials and data collection trials matched by recording each in a data collection notebook during the data collection session. This method served as a "double check" against the software system's output during data cleaning and processing.

The jump-landing tasks (double leg and single leg jump-landings) for this study were chosen because they consist of both a horizontal and vertical landing phase. Previous work has compared biomechanics during both horizontal and vertical landing phases of jump-landing tasks. The majority of previously reported differences between individuals with a PTA and controls are during *horizontal* landing phases, in which individuals are moving forward. These differences are thought to be attributed to greater magnitudes of posteriorly directed ground

reaction forces during horizontal landings, requiring greater internal knee extension moment to decelerate the limb and prepare for subsequent vertical movement.[23,26] Additionally, Edwards et al. (2012) demonstrated that peak patellar force is higher during the horizontal deceleration phase versus vertical phase of a landing task.[158] Furthermore, Cruz et al (2013) found that a forward vertical jump-landing from a box elicited greater hip and knee flexion, external knee flexion and abduction moments, external hip adduction moment, and anterior tibial shear force than drop-landing and drop vertical jump tasks.[220] Additionally, the forward jump-landing task is validated for clinical assessments of landing biomechanics.[221] Therefore, utilizing a forward jump-landing task, which incorporates a horizontal and vertical phase, placed the desired demands on the participant that are sought to evaluate differences between individuals at differing stages of tendon pathology.

The participants were fully oriented to the jump-landing task procedures. For the double-limb jump-landing task, participants performed five trials from a 30-centimeter box that was positioned 50% of the participant's height from the front edge of the force plates.[220,221] A total of five double-limb jump-landing trials were collected, and the middle three trials were averaged for data analysis. If one of the middle three trials was not successful, a subsequent trial was utilized for analysis. A successful double-limb jump-landing trial required the participant to leave the box with both feet at the same time, land on the force plates, and jump straight up in the air as high as possible (maximal vertical jump), and then land on two limbs back on the force plate.

For the single-limb jump-landing task, participants performed three trials over a six-inch tall hurdle starting at a distance 50% of the participant's height from the front edge of the force plates. A total of three single-limb jump-landing trials were collected on each limb, and all trials

were averaged for data analysis. The participant began each trial by standing on the limb being tested. A successful single-limb jump-landing trial requires that the participant clear the hurdle, land with the test limb on the force plate, jump straight up as high as possible (maximal vertical jump), and then land on two limbs back on the force plate.

Pain Assessment

Following the completion of the biomechanics assessment, the participant was escorted to the other side of the Sports Medicine Research Lab where the HUMAC Norm Dynamometer was located (~15 feet away). Retro-reflective markers on the shank, thigh, and hips were removed so as not to interfere with the intervention testing procedures. Next, participants performed the single-limb decline squat and were asked to report any pain that presented during this task on the NRS scale and pain map using the exact procedures described above during the screening session. The SLDS was performed on the involved limb only

Intervention Protocol

For the intervention protocol, the participant was positioned in the HUMAC chair at the exact same chair and body position that was used and recorded during the screening session (described above). Once the participant was appropriately positioned, the PI stepped out of the laboratory to another location within Fetzer Hall where she was unable to hear or see any constructs related to intervention delivery so that she remained blinded to the intervention (isometric or sham-control) for each session. Blinding of the PI was chosen in order to remove any potential bias that the PI's knowledge of the condition may have on processing and analysis of post-intervention pain and biomechanical assessments. A trained research assistant delivered the intervention. The intervention protocols were matched for time and rest. This involved timing the contraction (isometric condition) / sham TENS (sham-control condition) period (45 seconds)

and the rest period (2 minutes). The research assistant also ensured that the participant remained within +/- 5% of the target 70% MVIC throughout the 45-second isometric contraction by real-time observation of the participant's visual feedback (HUMAC Norm, CSmi, Stoughton, MA) screen during the contraction. The following details outline the procedures for the isometric and sham-control conditions.

Isometric Condition

The isometric condition protocol was selected based on the work by Rio et al. (2015), which demonstrated acute reductions in pain and improvements in quadriceps strength.[17] The participant performed five sets of a 45-second isometric contraction at 70% of the maximum voluntary isometric contraction (MVIC) that was obtained and calculated during the screening session. All participants were provided with the same standard instructional script: *"During this session, you will see a green line displayed on the screen, which represents the amount of force that you produce with your quadriceps contraction during the test. You will see two purple lines with a dark zone in between them, which are centered around 70% of the maximum value of the muscle contraction you were able produce during your first testing session. Your goal is to maintain your muscle contraction so that the green contraction line stays as close to the middle of the dark zone and does not go above or below the purple lines, for the entire duration of the repetition. If the green line goes above or below the purple lines, the clock will stop until the green line returns to the dark zone. Focus on using your quadriceps muscle contraction to smoothly control the green line. You will maintain this muscle contraction for 45-seconds, followed by 2 minutes of rest. We will repeat this same sequence 5 total times."*

During the 45-second contraction, the participant was provided with visual biofeedback on a computer screen positioned directly in front of the chair. Visual biofeedback was provided

via an internal feature within the HUMAC Norm software, including a real-time display of the 70% MVIC target line (green) and +/- 5% error lines (purple) around the 70% MVIC line. The display moved in real-time from left-to-right during the 45-second contraction so that there was no delay in what the participant is able to see. The participant was instructed to produce a level of isometric quadriceps contraction that maintained the isometric torque output line as close the target line as possible and always between the two error lines. Following each 45-second isometric contraction, the participant had 2-minutes of rest, during which his limb remained positioned at 60° knee flexion but no contraction performed. Following 5 consecutive sets, the participant completed the isometric intervention and was be un-strapped from the HUMAC chair.

Sham-Control Condition

For the sham-control condition, participants were positioned in the identical position that used during the screening and isometric condition sessions. However, during this condition there was no voluntary quadriceps contraction; instead a sham transcutaneous electric nerve stimulator (TENS) unit (Empi Select™ TENS Device; Empi, Inc., St. Paul, MN, USA) was utilized as a sham intervention. The rationale behind utilizing this type of sham-control condition was to avoid any participant-bias that the intervention condition (isometric condition) was the experimental condition, as this perception could bias their performance on the post-intervention SLDS pain and biomechanics assessments.

Two (2" x 2") electrodes were placed on either side (medial and lateral) of the patellar tendon (not on the tendon) on the test limb and connected via two lead wire to the sham TENS unit that was held and controlled by the trained research assistant. Instructions were given to the participant prior to the beginning of the sham-control condition; the same instruction script was

used for each participant. The instructions specified that, *"A surface electrode has been placed on either side of your patellar tendon. I will turn on the stimulation unit to emit a stimulus to your patellar tendon. This is a special sub-sensory stimulation treatment, so you will not feel anything during this period as the stimulus is set at a very low, non-detectable threshold. Please remain still during this 45-second period, letting your leg rest passively in the machine without contracting your leg muscles. After the 45-second period, the stimulation unit will be turned off and you will have a 2-minute rest period. We will repeat this same treatment/rest sequence 5 total times."*

The same intervention parameters were utilized for the sham-control condition (5 sets of a 45-second repetition with 2-minutes of rest between each repetition).

Post-Intervention Protocol Reassessments

Immediately following the conclusion of the intervention protocol, participants repeated the same pre-intervention protocol assessments using identical methodology as described above in the following order:

1. Pain assessment (SLDS board next to HUMAC Norm Dynamometer)
2. 3-D Biomechanics Assessment (Motion capture area)

Following the completion of Session 2, participants were reminded of their Session 3 schedule, ensuring that a date and time has been scheduled appropriately. Sessions 2 & 3 were conducted 7-10 days apart (wash-out period) in order to ensure that there were no carry-over effects from the intervention condition performed during Session 2. Participants were asked to maintain their normal training and competition parameters during this period. Participants were reminded via phone and/or email 24 hours prior to the Session 3 date to enhance compliance and minimize risk of no-show. Approximately 7-10 days following Session 2, participants returned to

the Sports Medicine Research Laboratory for Session 3. Identical procedures were carried out, with the exception of the assignment for the intervention protocol. The participants performed the intervention condition that they did not perform during Session 2.

Instrumentation

Three-Dimensional Motion Capture Biomechanical Data Collection Instrumentation

Three-dimensional marker coordinate data were collected using an optoelectric retroreflective ten-camera motion capture system (Bonita 10, Vicon Motion Systems, Centennial, CO), sampling at 120 Hz. The ten-camera system is oriented around a 2x2x2 meter capture area centered around two floor-embedded force plates (Type 4060-10, Bertec Corporation, Columbus, OH) which was utilized to capture ground reaction force data, sampling at 1200 Hz.

Prior to data capture, the capture area was calibrated using a 5-marker wand to establish marker identification within the world, as well as to set the volume origin for the world axes. The laboratory's global axis system is defined as follows: positive x-axis directed anteriorly (anterior-posterior axis), positive y-axis directed left (medial-lateral axis) and positive z-axis directed superiorly (super-inferior axis) with respect to the participant in the capture area. Marker coordinate data was streamed to a computer (Dell Precision T5610, Round Rock, TX) and saved within Vicon Nexus v1.7.1 motion capture software via a Vicon Ultranet MX Controller (Vicon Systems, Centennial, CO). The embedded Bertec force plates were calibrated to the laboratory's global coordinate system (see above) that is delineated during marker system camera calibration. Analog force plate data was sampled at 1200 Hz and transmitted through an A/D board which is interfaced and synchronized with the Vicon Nexus motion capture software. Marker coordinate data and ground reaction force data were then saved within Vicon Nexus to allow real-time marker identification.

Finally, all marker coordinate (kinematic) and ground reaction force (kinetic) data were transferred into The Motion Monitor software (Innovative Sports Training, Chicago, Illinois) to build three-dimensional link-segment models for biomechanical data analysis and reduction.

Physical Activity Monitoring Data Collection Instrumentation

The primary outcome variable that will be collected during the one-week monitoring period was steps-per-day and MVPA minutes/day. The ActiGraph GT9X Link accelerometer (3.5 x 3.5 x 1cm; 14 grams) measures accelerations in the range of +/- 8g and at a sampling frequency of 30-100Hz. For this study, the accelerometer was set in raw data capture mode to sample raw acceleration data at 30 Hz during daily wear periods.

DATA PROCESSING & REDUCTION

Laboratory Biomechanics: Three-Dimensional Motion Capture Data

Marker Identification & Processing

Following marker identification and labelling, both marker data and ground reaction force data (synchronized) were saved as a .c3d file and exported. Next, the .c3d file was imported into *The Motion Monitor Software* for construction of a link-segment model for each trial of each participant. Lower extremity segments were modeled as rigid bodies using three non-collinear individual markers for each segment, as follows:

- Left foot: rigid cluster of four markers
- Right foot: rigid cluster of three markers
- Left shank: rigid cluster of four markers
- Right shank: rigid cluster of three markers
- Left thigh: rigid cluster of four markers
- Right thigh: rigid cluster of three markers

- Sacrum/pelvis: left ASIS, right ASAI, L5-S1 marker

Joint Center Calculations

Within *The Motion Monitor* software, ankle, knee, and hip joint center coordinates were defined based on the adjacent segments, described above, around each respective joint bilaterally, taken during the static trial. The ankle joint center was defined as the centroid between the medial and lateral malleoli, representing the end-points of the shank segment. The knee joint center was defined as the centroid between the medial and lateral condyles, representing the end-points of the thigh segment. Hip joint center coordinates were estimated from the coordinates of the L5-S1, right ASIS, and left ASIS markers using the Bell method.[222]

Kinematic Calculations

Joint angles were defined based on the position of the distal segment relative to the proximal segment using a Cardan angle sequence in the following order of rotation: sagittal (y-axis), frontal (x-axis'), and transverse (z-axis'). Kinematic variables of interest for this study included: bilateral hip flexion (-)/extension (+), hip adduction (+)/abduction (-), knee flexion (+), knee varus (+)/valgus (-), and ankle plantarflexion (+)/dorsiflexion (-).

Kinetic Calculations

Ground reaction force data and processed segment data were used to calculate net internal sagittal and frontal plane knee and hip joint moments using the inverse dynamics procedures described and well-accepted in the biomechanics literature.[146] Net internal sagittal plane knee and hip moments characterize the forces acting about the knee and hip medial-lateral axes of rotation, respectively (y-axis). Net internal frontal plane knee and hip moments characterize the forces acting about the knee and hip anterior-posterior axes of rotation, respectively (x-axis). Vertical ground reaction force (vGRF) were calculated as the vertical

component of the ground reaction force vector that aligns with the world z-axis.[146] Patellar tendon force (F_{PT}) was estimated using previously defined methodology of Nisell and Ekholm (1985),[160] through with F_{PT} is calculated by dividing the internal knee extension moment by the patellar tendon moment arm via the equation (Equation 1):

$$F_{PT} = \frac{knee\ extension\ joint\ moment}{PT\ moment\ arm}$$

(Equation 1)

where the patellar tendon moment arm was calculated as a function of the knee joint angle as described by Herzog and Read (1993).[144]

Data Reduction

All segmental kinematic and kinetic data, as well as ground reaction force and patellar tendon force data were processed in The Motion Monitor software. Data was filtered using a fourth-order low-pass Butterworth filter with a 20Hz cutoff frequency. All kinematic data was interpolated and synchronized with the raw 1200 Hz ground reaction force data. Next, filtered segmental biomechanical data and ground reaction force and patellar tendon force data was exported from The Motion Monitor software into a custom MatLab software program (MatLab R2017b, The MathWorks, Inc., Natick, MA) for further data inspection and reduction.

Phase Identification

Biomechanical variables for each limb were evaluated across the entire stance phase. The stance phase was defined as the time from initial ground contact (IC), or the time point when the vertical ground reaction force (vGRF) exceeds 10N, until toe-off (vGRF <10 N).[223] The stance phase was further divided into the *loading* phase (IC through peak knee flexion position) and the *propulsive* phase (peak knee flexion position to toe-off). Peak knee flexion position was defined as the time point when the knee reached its maximum flexion angle during the stance phase.

Dependent Variable Calculation

Aim 1: Kinematic (Table 1.1, RQ 1.1) and kinetic (Table 1.1 RQ 1.2 – 1.4) variables for Aim 2 were analyzed as continuous normalized waveforms during the stance phase of the landing tasks (Kuenze et al. 2014).[224] For each kinematic and kinetic dependent variable, the within-group mean values were interpolated and normalized over 202 data points (knots) over the stance phase of the middle three landing trials for each task using a cubic spline filter. These data points represent 0% - 100% of the stance phase of the task (initial ground contact through toe-off, respectively). Each knot was calculated as the mean value of the derived knots from each of the middle three trials (t_1, t_2, t_3) of each respective task used for analysis (Equation 2):

$$knot_{i\ldots202} = \frac{knot_{it1} + knot_{it2} + knot_{it3}}{3}$$

(Equation 2)

Limb symmetry indices (LSI) were calculated for all dependent biomechanical variables using the following equation (Equation 3):

$$LSI = \left(\frac{involved\ limb}{uninvolved\ limb}\right)$$

(Equation 3)

where values <1 represent lower magnitude variable on the involved / matched healthy control limb compared to the uninvolved / contralateral healthy control limb, and value > 1 represent greater magnitude variable on the involved / matched healthy control limb compared to the uninvolved / contralateral healthy control limb. The same continuous waveform analyses were utilized to compare LSI for dependent variables across the stance phase of each task.

Aim 3: Kinematic variables (Table 1.3, RQ 3.1) were calculated at initial ground contact, peak, and displacement across the loading phase of the landing task for the involved PTA and matched healthy control limbs. Net internal joint moment variables (Table 1.3, RQ 3.2) were

calculated across the first 50% of the loading phase of the landing task for the involved PTA and matched healthy control limb. Finally, vGRF and F_{PT} variables (Table 1.3, RQs 3.3 and 3.4) were assessed for their peak magnitude and impulse across the loading phase.

Real-World Physical Activity: Cumulative External Load Monitoring

Data Processing & Reduction

Data was processed and analyzed using ActiLife v6.0.0 software (ActiGraph Corporation, Pensacola, FL), which is the actigraphy data analysis software platform of ActiGraph. The Sports Medicine Research Laboratory is equipped with one desktop and six laptop licensure keys for ActiLife processing.

Wear time validation was performed based on the Choi et al. (2011) algorithms,[225] whereby the following parameters were selected within ActiLife software:

a) Minimum wear time per day: 480 minutes
b) Minimum days of valid wear time: 4 days
c) Minimum weekdays of valid wear time: 3 days
d) Minimum weekend days of valid wear time: 1 day.

The following processing settings were selected within ActiLife software:

a) Energy expenditure: Freedson VM3 Combination (2011)
b) METs: Freedson Adult (1998)
c) Cut Points and MVPA: Freedson Adult VM3 (2011)
d) Bouts: checked
e) Sedentary Analysis: checked
f) Exclude Non-Wear Time: checked

The number of steps-per-day is calculated within ActiLife software based on the vertical acceleration data measured with the GT9X Link monitor. GT9X Link files were uploaded to ActiLife software platform and converted into 60-second epoch '.agd' files for analysis of count data. Data is cleaned and scored within the ActiLife software. Data files were exported as .csv files with the following data: header definitions, desktop summary (summary data for each participant), and wear time validation (wear periods for each participant).

Dependent Variable Calculation

Aim 2: The dependent variable 'average steps-per-day' and 'average MPVA/day' (Table 1.2, RQ 2.1) were extracted from ActiLife software for the one-week monitoring period, and normalized to the number of wear days for each participant. The dependent variable 'cumulative patellar tendon load' (cF_{PT}) (Table 1.2, RQ 2.2) was calculated based on the methodology of Maly et al. (2013) that estimated cumulative knee adductor load per day based on laboratory biomechanical assessments and steps-per-day$_{MVPA}$.[39] The number of steps-per-day$_{MVPA}$ was divided by two, since the accelerometer captures steps for both limbs. The following equation (equation 4) was utilized:

$$\textbf{cF}_{\textbf{PT}}\textbf{ load estimation: } \int_a^b F_{PT}(t) * dt \times \left(\frac{steps_{MVPA}}{day}\ for\ test\ limb\right)$$

(Equation 4)

where cF_{PT} is the cumulative patellar tendon force per day estimation; $F_{PT}(t)$ is the mean patellar tendon force (F_{PT}) across the five jump-landing trials at time (t) from a) initial ground contact, to b) toe-off.

STATISTICAL ANALYSIS

Means, standard deviations, medians, interquartile ranges, and 95% confidence intervals were calculated for all demographic and questionnaire data where appropriate. Alpha was set *a*

priori at $\alpha < 0.05$ for all statistical analyses. Normality was assessed for all dependent variables using the Shapiro-Wilk test and normal Q-Q plot inspection. All statistical analyses were completed in SPSS v22 (IBM Inc., Armonk, New York, USA).

Aim 1: *To ascertain the impact of symptomatic PTA and asymptomatic PTA on lower extremity landing kinematics and kinetics.*

To evaluate baseline biomechanical characteristic between the three study groups, 95% confidence interval waveforms across the stance phase for each movement task were plotted.[224] Each variable curve was evaluated for areas where the group curve did not overlap in order to determine if there was an interaction between the three study groups (SYM-PTA, ASYM-PTA, CON) and each kinematic and kinetic dependent variable (Table 1.2). Average Cohen's *d* effect sizes and mean differences with standard deviations were calculated for all areas of non-overlap by taking the average of all data points included in a given area of non-overlap.

Aim 2: To ascertain the impact of symptomatic PTA and asymptomatic PTA on cumulative load volume during a one-week monitoring period.

Descriptive statistics were calculated for all variables of interest, including means, standard deviations, and 95% confidence intervals. A one-way analysis of variance (ANOVA) was conducted to determine if there were differences in cumulative load volume variables between the three study groups (SYM-PTA, ASYM-PTA, CON). Post-hoc testing using Bonferroni post-hoc adjusted *t*-tests for pairwise comparisons of means for each dependent variable were performed for significant findings from each ANOVA model.

Aim 3: *To investigate whether an acute isometric patellar tendon loading exercise protocol changes lower extremity landing kinematics and kinetics in individuals with symptomatic and asymptomatic PTA.*

Descriptive statistics were calculated for all variables of interest, including means, standard deviations, and 95% confidence intervals. We established four *a priori* comparisons to investigate both within-group and between-group effects of each intervention condition (isometric and sham-TENS). A 2x2 mixed-model repeated measures analysis of variance (ANOVA) (group: 2 levels (SYM-PTA, ASYM-PTA); Treatment: 2 levels (isometric and sham-control) on change scores for each biomechanical dependent variable for the involved limb from pre- to post-intervention was utilized to evaluate the effects of each condition on dependent variables for the two groups (SYM-PTA, ASYM-PTA). Change scores (Δ) were calculated for each biomechanical dependent variable using the following equation (Equation 5):

$$Change\ score\ (\Delta) = mean_{post} - mean_{pre}$$

(Equation 5)

Cohen's *d* effect sizes for each treatment condition were calculated using the following equation, when appropriate (Equation 6):

$$Cohen's\ d_{t_i} = \frac{mean_{t_i post} - mean_{t_i pre}}{pooled\ standard\ deviation_{t_i}}$$

(Equation 6)

where t_i is the treatment condition (either isometric or sham-control).

Post-hoc testing using Bonferroni post-hoc adjusted *t*-tests for pairwise comparisons of means for each dependent variable will be performed for significant findings from each ANOVA model.

POWER ANALYSIS

An *a priori* power analysis was completed using G*Power (Version 3.1.9.2; Kiel University) to determine the sample size needed to detect significant differences between symptomatic and asymptomatic individuals, as well as between individuals with and without

patellar tendon structural abnormality, based on previous literature evaluating these populations. The power analysis was run for key biomechanical and cumulative external load variables of interest and for exercise-response variables. This *a priori* sample power calculation was completed with $\alpha_{two\text{-}tailed} = 0.05$, $1\text{-}\beta = 0.80$ and $1\text{-}\beta = 0.90$, and effect sizes as listed below. Cohen's d effect sizes are classified as weak (<0.2), small (0.21-0.50), medium (0.51-0.8), and large (>0.80).[226]

To-date, no studies have been conducted to examine the effect of knee extension-based exercise on biomechanical outcome measures in individuals with patellar tendinopathy (symptoms or structural pathology). Edwards et al. (2010) compared biomechanical variable between independent groups (PTA versus no-PTA) cross-sectionally and demonstrated moderate-to-large differences between groups in peak vGRF, peak knee and hip flexion angle, and knee and hip flexion displacement.[23] Additionally, previous studies using differing intervention paradigms (visual and verbal biofeedback) have shown moderate (d=0.78) to large (d=1.85) effects on biomechanical variables (peak vGRF and knee flexion angle, respectively).[227–229] Rio et al. (2015) found a large effect (d=2.75) pre- to post-isometric loading protocol on SLDS pain.[17]

We chose to power this study off of our key biomechanical variable of interest. Based on this power analysis, a sample size of 15 participants in each group will allow the principal investigator to detect, at minimum, a 20% difference in biomechanical profiles between participants, with a power of at least 80% and $\alpha_{two\text{-}tailed} = 0.05$. A study sample with 15 participants in each of the three groups (SYM-PTA, ASYM-PTA, and CON) was determined to provide enough power to detect clinically meaningful differences in biomechanics.

CHAPTER 4: RESULTS

Specific Aim 1

To ascertain the impact of symptomatic PTA and asymptomatic PTA on lower extremity landing kinematics and kinetics.

Results

No significant differences in height and mass were observed between groups ($p > 0.05$), but the ASYM group was slightly older than the CON group (p=0.045). The VISA-P score was significantly lower in the SYM group compared to both the ASYM and CON groups (p<0.001), and the mean differences exceeded the MCID (13 points) for this subjective outcome measure.[204] There were no differences in VISA-P score between the ASYM and CON groups (p>0.05).

Double Limb Landing

Kinematics

Participants in the SYM group demonstrated lesser magnitude knee flexion angle than the CON group throughout the majority of the stance phase (8-76%, *d*: 1.14±0.12, MD: 15.83±2.71°). Participants in the ASYM group demonstrated lesser magnitude knee flexion angle than the CON group during the early (8-13%, *d*: 0.99 ± 0.04, MD: 7.99±0.39°; 21-24%, *d*: 1.01±0.01, MD: 11.11±0.32°) and late (74-94%, *d*: 0.96 ± 0.07, MD: 9.55 ± 1.13°) portions of the stance phase. There were no differences between the SYM and ASYM groups in sagittal plane knee angle.

Kinetics

Participants in the SYM group demonstrated lesser internal knee extension moment than the CON group during early stance (6.5-9%, *d*: 1.21± 0.08, MD: 0.04 ± 0.004 N*m[kg*m]$^{-1}$), as well as the ASYM group during mid-stance (38-56%, *d*: 1.17 ± 0.06, MD: 0.03 ± 0.001 N*m[kg*m]$^{-1}$). There were no differences between the ASYM and CON groups in internal sagittal plane knee moment.

There were no differences in vGRF between groups. However, the SYM group demonstrated less patellar tendon force during early stance (6-9%, *d:* 1.15 ± 0.15, MD: 0.85 ± 0.15 (BW)) than the CON group and during mid-stance (36-60%, *d*: 1.22 ± 0.08, MD: 0.66 ± 0.05 (BW)) than ASYM group. There were no differences between the ASYM and CON groups in patellar tendon force. Finally, participants in the SYM group had less knee power during early stance (6-9%, *d*: 1.24 ± 0.17, MD: 0.48 ± 0.16 J/s; 18.5-23%, *d*: 1.34 ± 0.13, MD: 0.17 ± 0.01 J/s) than the CON group and during early stance (20.5-25%, *d*: 1.14 ± 0.08, MD: 0.20 ± 0.01 J/s) than the ASYM group (Figure 6). There were no differences in knee power between the ASYM and CON groups.

Limb Symmetry Index

There were no differences in limb symmetry indices for VGRF, F_{PT}, or KP between any of the groups across the entire stance phase. Additionally, within each group, there were no areas during which the 95% CI did not overlap with 100, suggesting that there was no inter-limb asymmetry within each group.

Single Limb Landing

Kinematics

There were no differences in any sagittal or frontal plane kinematic variable at the hip or knee between any of the groups.

Kinetics

There were no differences in sagittal or frontal plane knee moments or sagittal plane hip moments between any of the groups. Participants in the SYM group demonstrated greater internal frontal plane hip abduction moment than the CON group for the majority of the stance phase (23-79%, *d*: 1.10± 0.07, MD: 0.04 ± 0.003 N*m[kg*m]$^{-1}$). Participants in the ASYM group demonstrated greater internal frontal plane hip abduction moment than the CON group during the late stance phase (84-91%, *d*: 1.12± 0.085 MD: 0.02 ± 0.001 N*m[kg*m]$^{-1}$).

There were no differences in VGRF or knee power between any of the groups. Participants in the SYM group demonstrated lesser patellar tendon force than the CON group during early stance (16-19%, *d*: 1.08 ± 0.03, MD: 0.90 ± 0.05 (BW)).

Summary

In summary, the overall landing profile of the SYM group during the double-limb landing task was consistent with our hypotheses that these individuals would demonstrate patterns of under-loading of the involved limb due to symptoms. On average, this group demonstrated decreased sagittal plane motion compared to both CON and ASYM groups for the majority of the stance phase, and also demonstrated lesser knee loading (internal knee extension moment, patellar tendon force, and knee power) than the CON group during the early loading response phase of the landing task. Interestingly, the landing profile of the ASYM group was in contrast to our hypothesis of an over-loading profile, as noted in previous literature.[23] While the ASYM

group did demonstrate reduced sagittal plane motion at early- and late- phases of stance compared to the CON group, there were no other significant differences in their profiles compared to the CON group, and no signs of over-loading for any kinematic, kinetic, or energetic variable. Finally, there were minimal differences between groups during the single-leg landing task.

The results of this aim support the hypothesis that athletes with symptomatic patellar tendinopathy tend to unload their involved limb during critical periods of a landing task where tissue loads are greatest. However, there was no evidence of over-loading in asymptomatic individuals. Despite lacking pain, these individuals may be demonstrating early signs of under-loading that need to be targeting through controlled loading programs to maximize tissue capacity. The results of this study provide further evidence to support an individualized approach to movement assessment and retraining in individuals at different stages of the continuum of tendinopathy

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Figures

The following outline all of the waveforms for the *double-limb landing task.*

Figure 4.1: Mean and 95% confidence interval waveforms for involved limb knee kinematic variables during the double-limb jump landing task

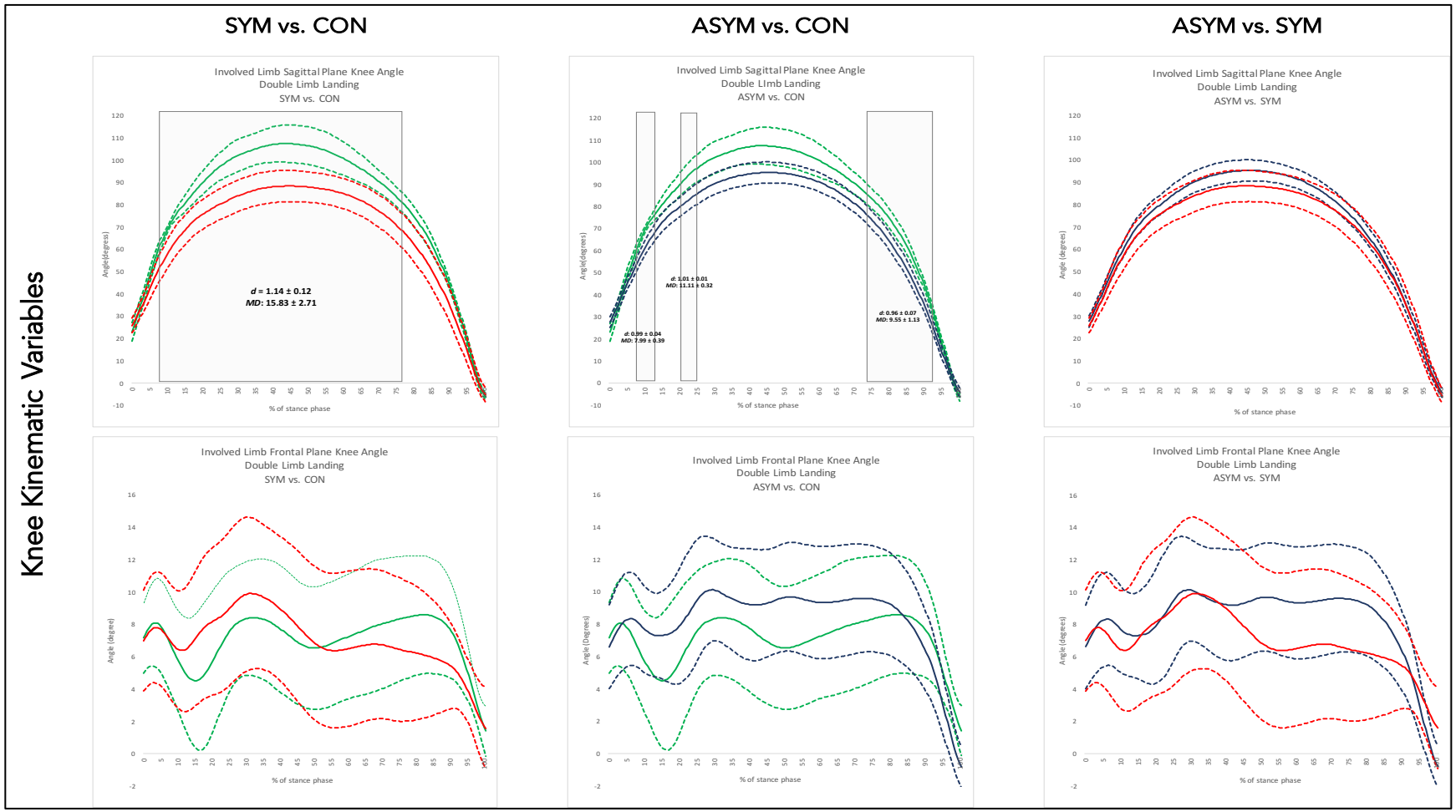


Figure 4.2: Mean and 95% confidence interval waveforms for involved limb hip kinematic variables during the double-limb jump landing task

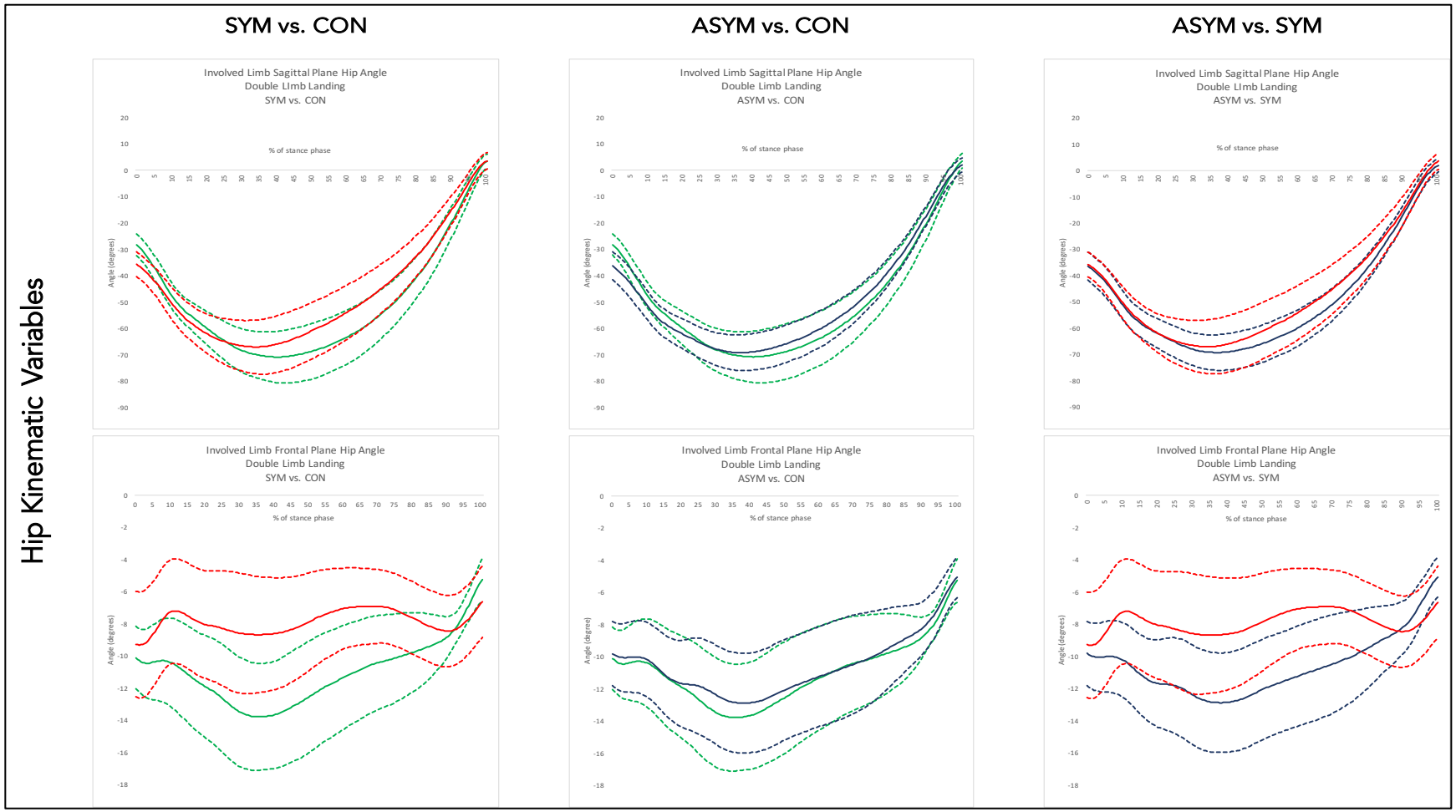


Figure 4.3: Mean and 95% confidence interval waveforms for involved limb knee moment variables during the double-limb jump landing

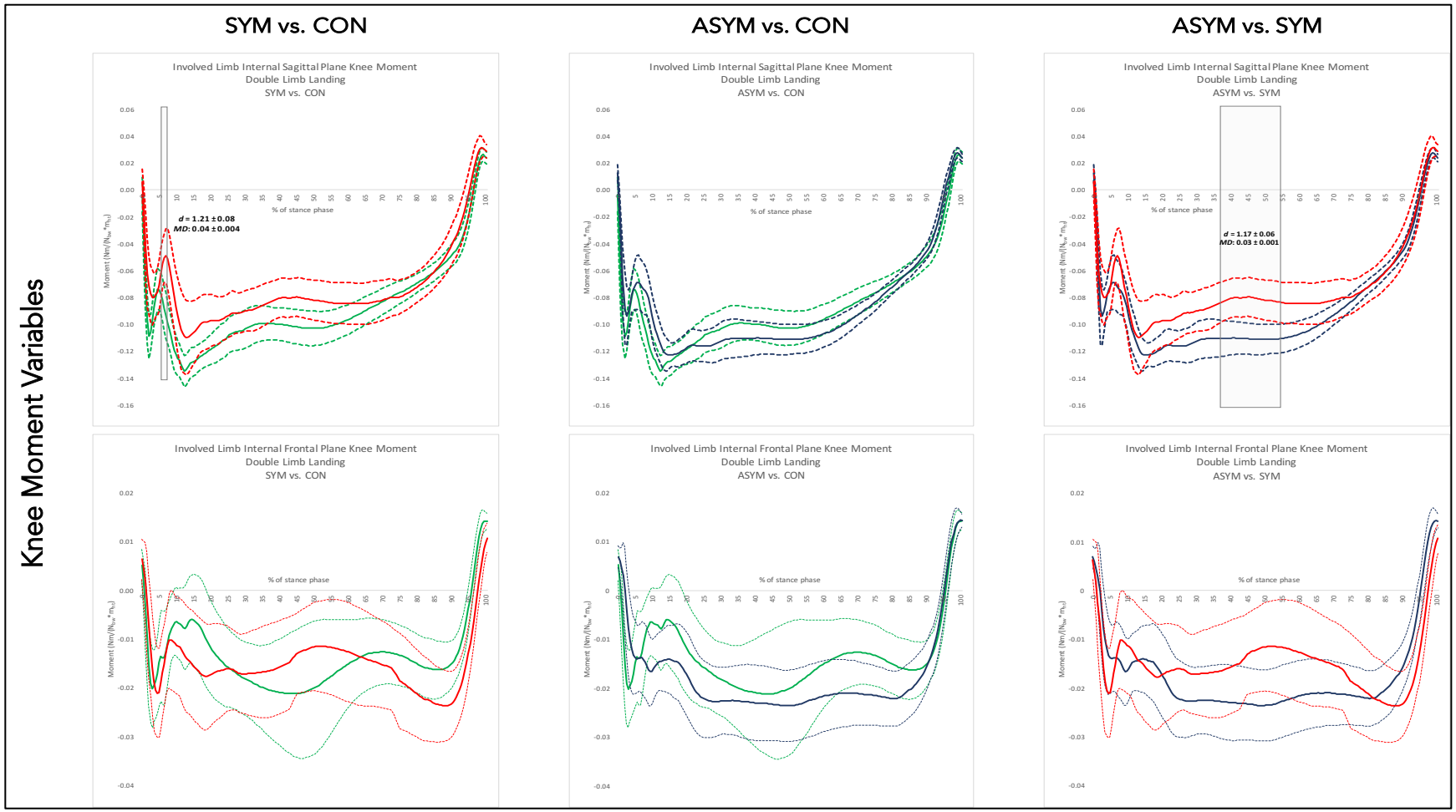


Figure 4.4: Mean and 95% confidence interval waveforms for involved limb hip moment variables during the double-limb jump landing

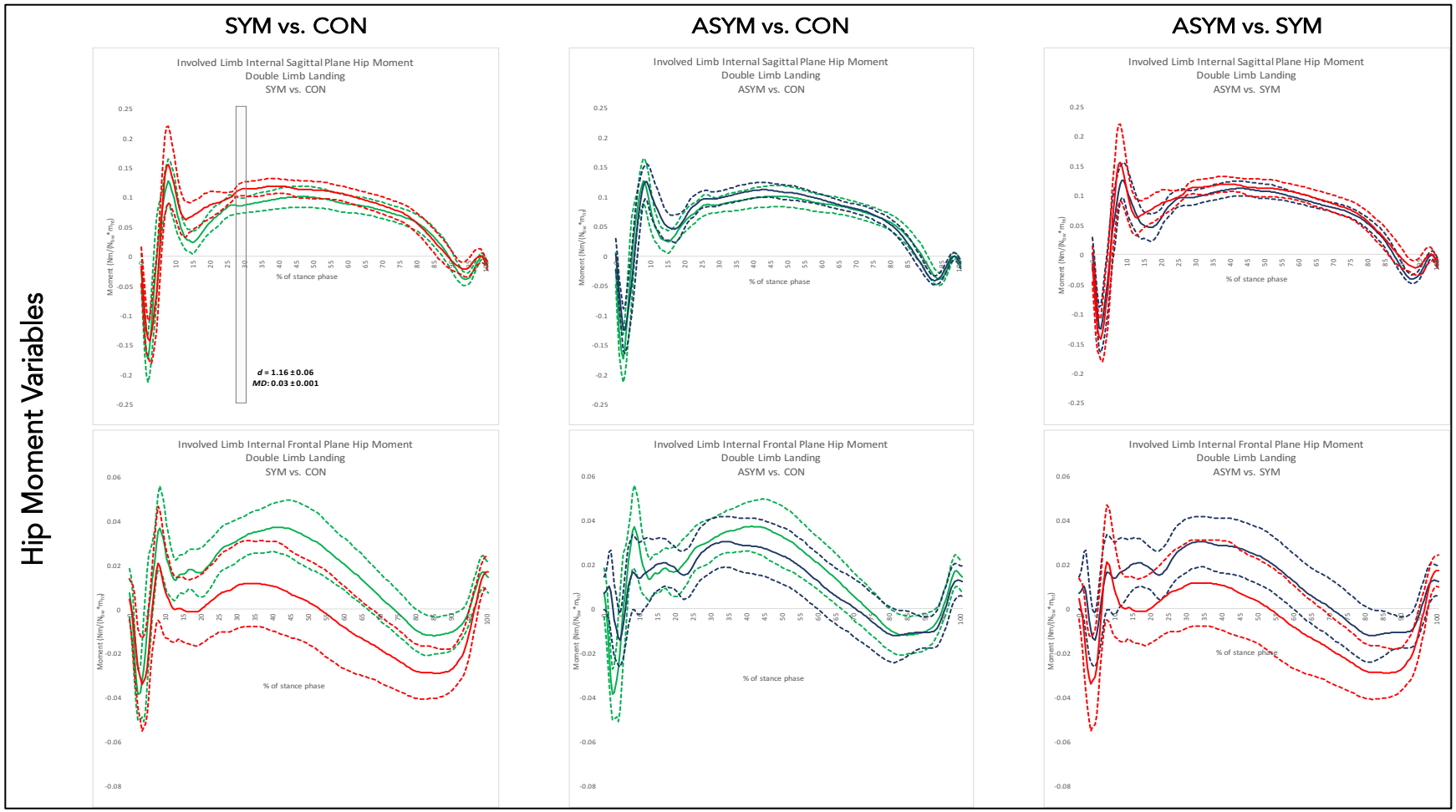


Figure 4.5: Mean and 95% confidence interval waveforms for involved limb vertical ground reaction force (VGRF) and patellar tendon force (PTF) variables during the double-limb jump landing

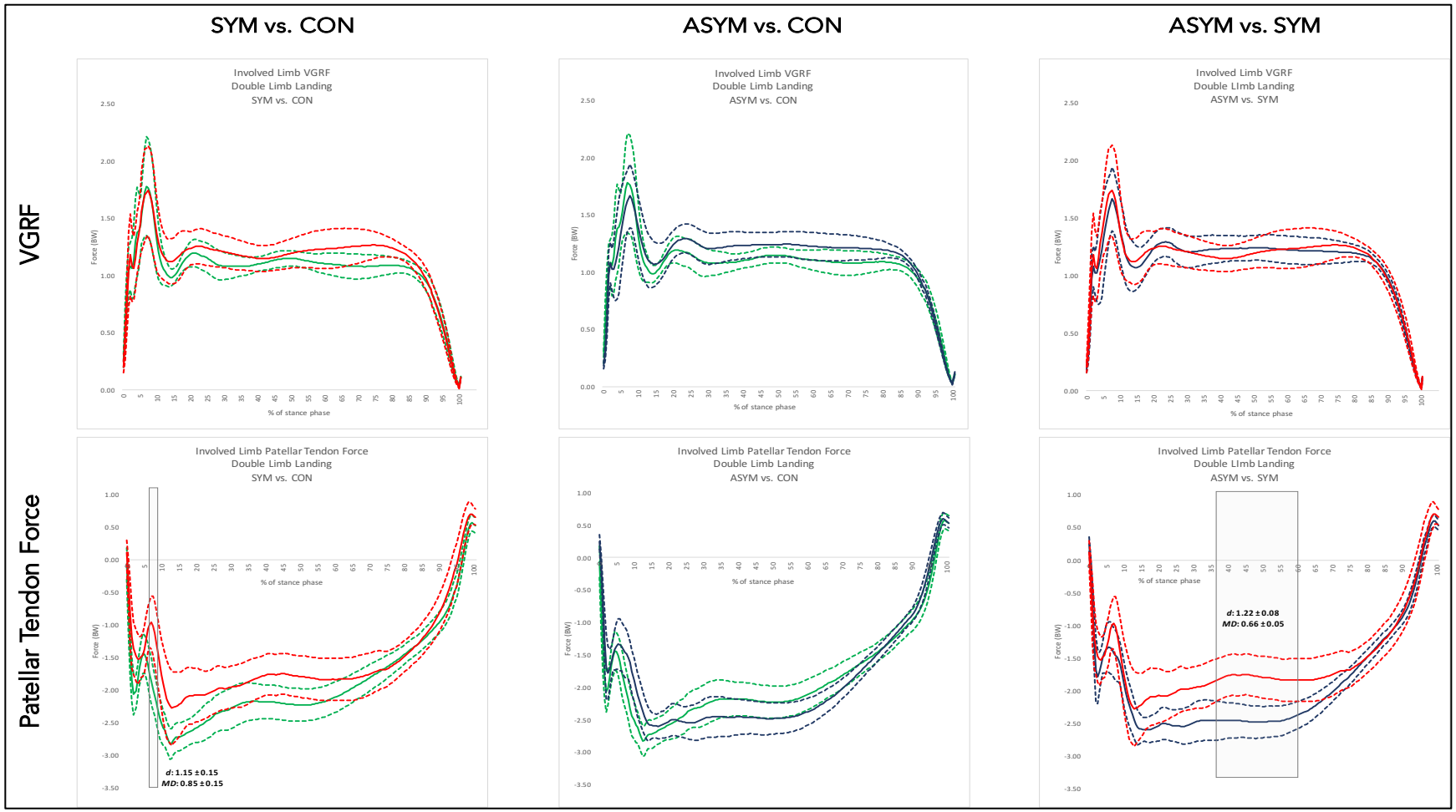


Figure 4.6: Mean and 95% confidence interval waveforms for involved limb knee power during the double-limb jump landing

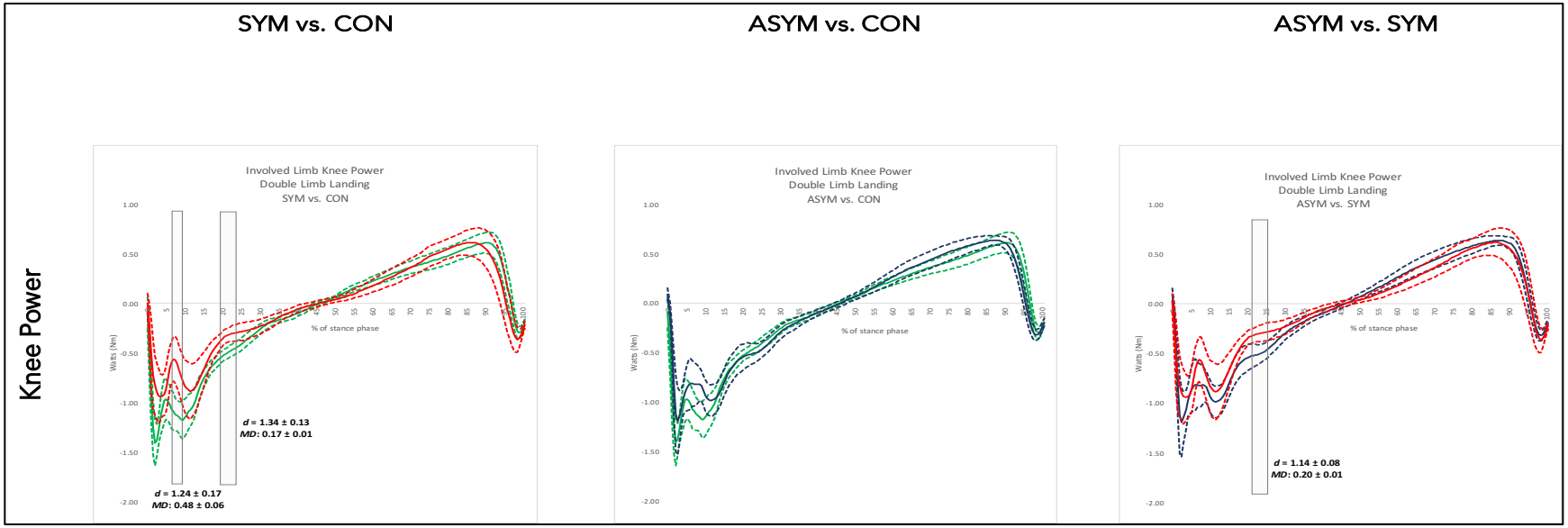


Figure 4.7: Mean and 95% confidence interval waveforms for vertical ground reaction force (vGRF) limb symmetry indices during the double-limb jump landing.

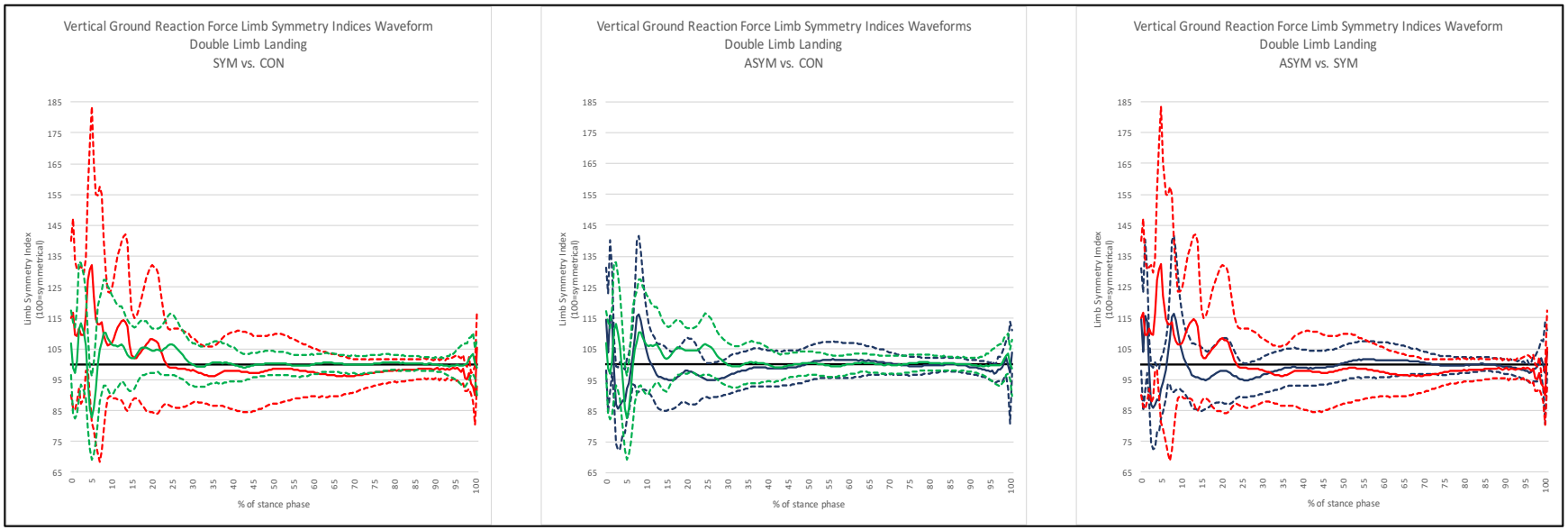


Figure 4.8: Mean and 95% confidence interval waveforms for internal knee extension moment limb symmetry indices during the double-limb jump landing.

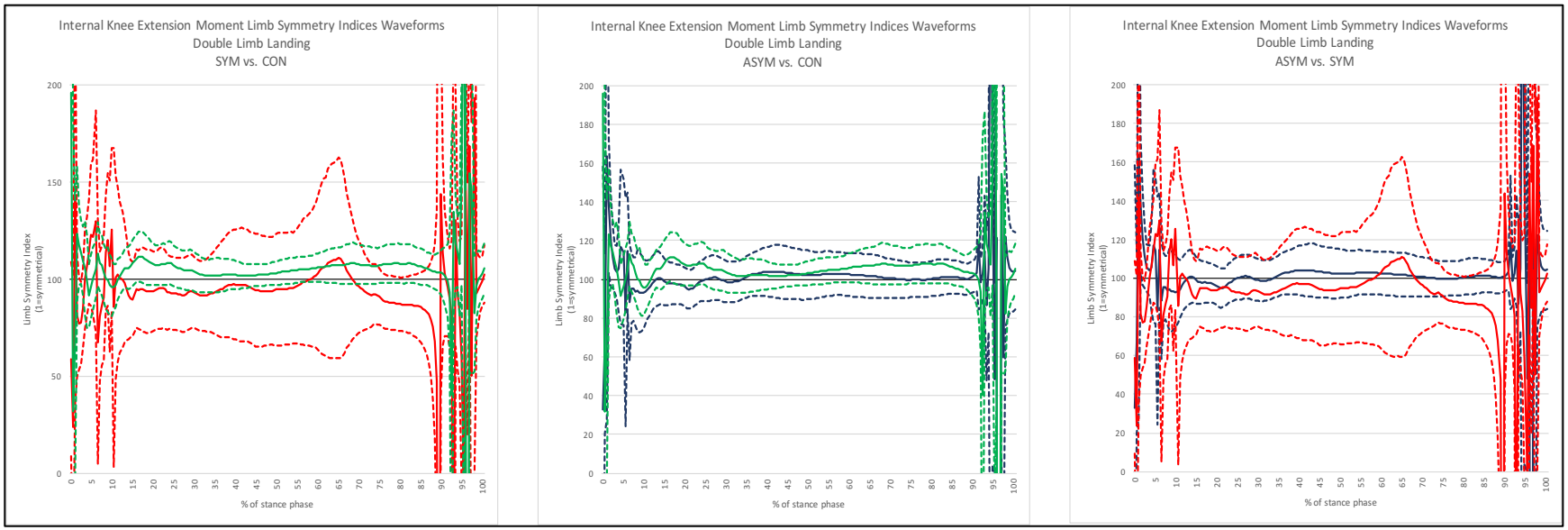
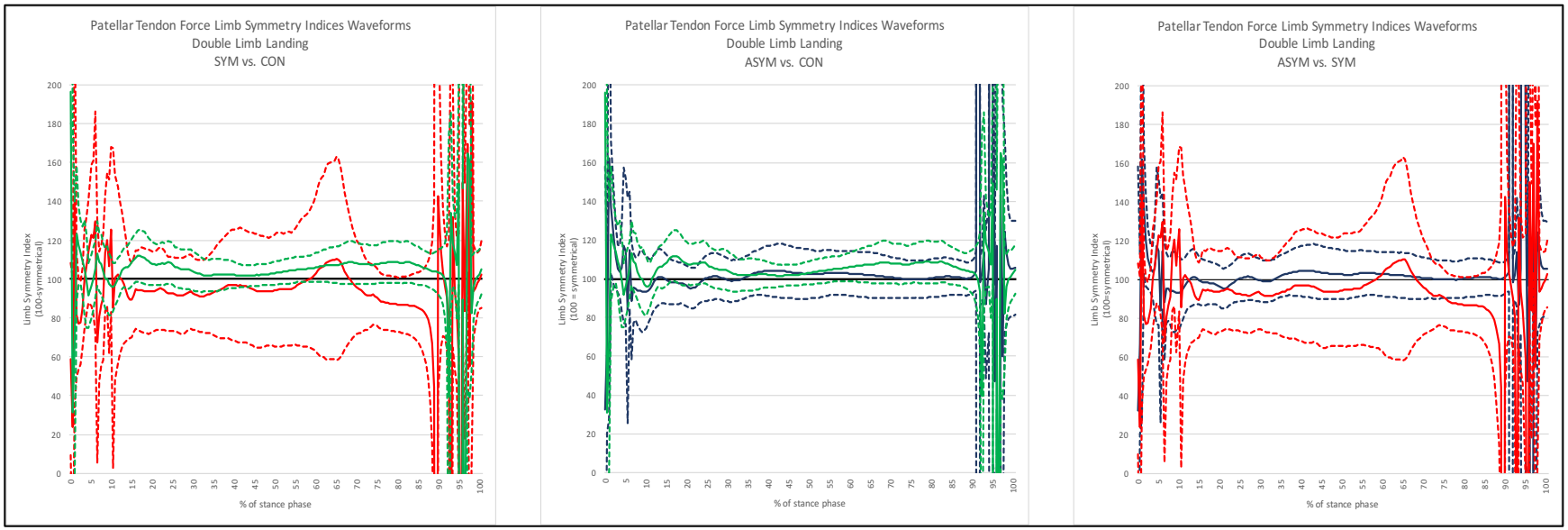


Figure 4.9: Mean and 95% confidence interval waveforms for patellar tendon force limb symmetry indices during the double-limb jump landing.



The following outline all of the waveforms for the *single-limb landing task.*

Figure 4.10: Mean and 95% confidence interval waveforms for involved limb knee kinematic variables during the single-limb jump landing task

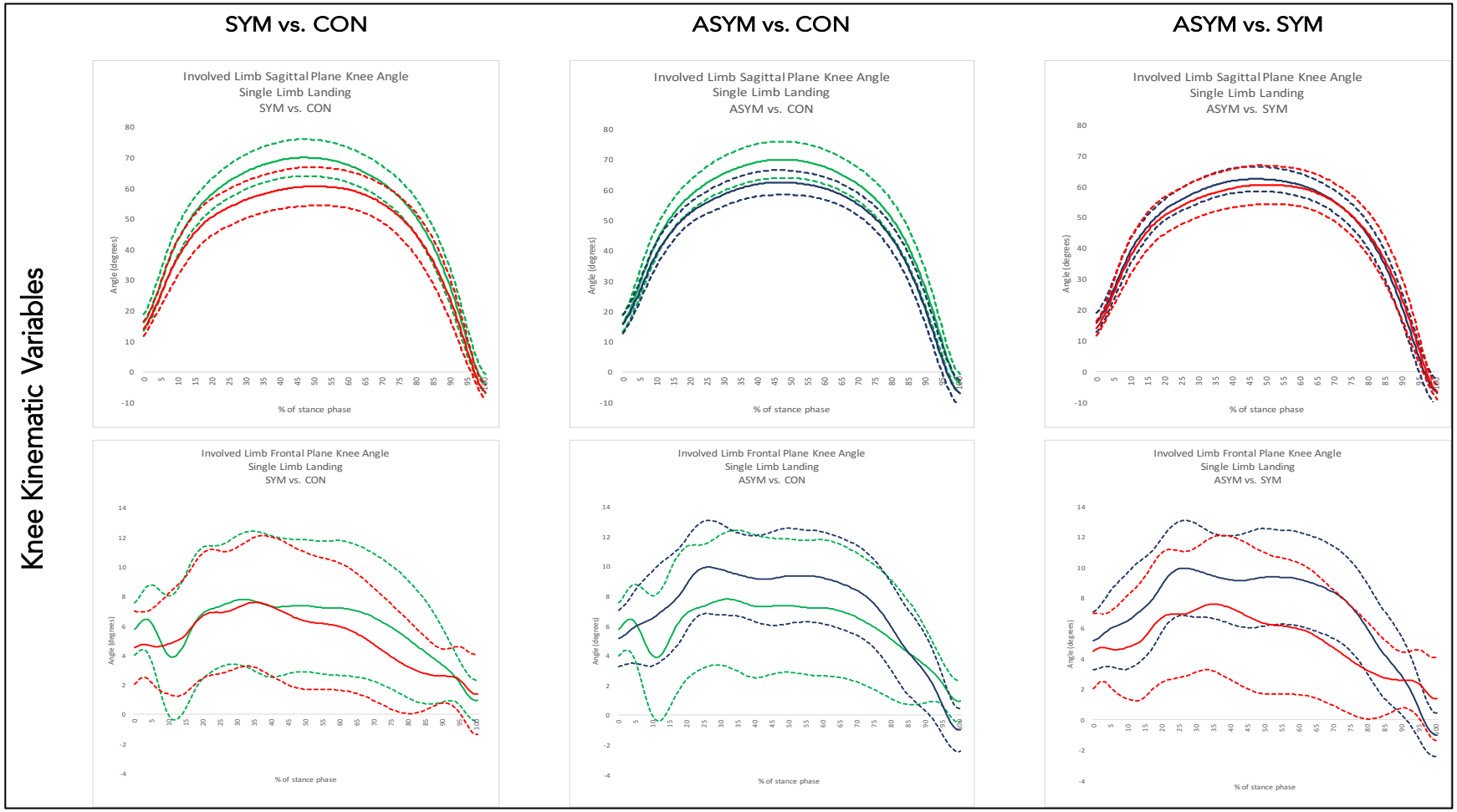


Figure 4.11: Mean and 95% confidence interval waveforms for involved limb hip kinematic variables during the single-limb jump landing task

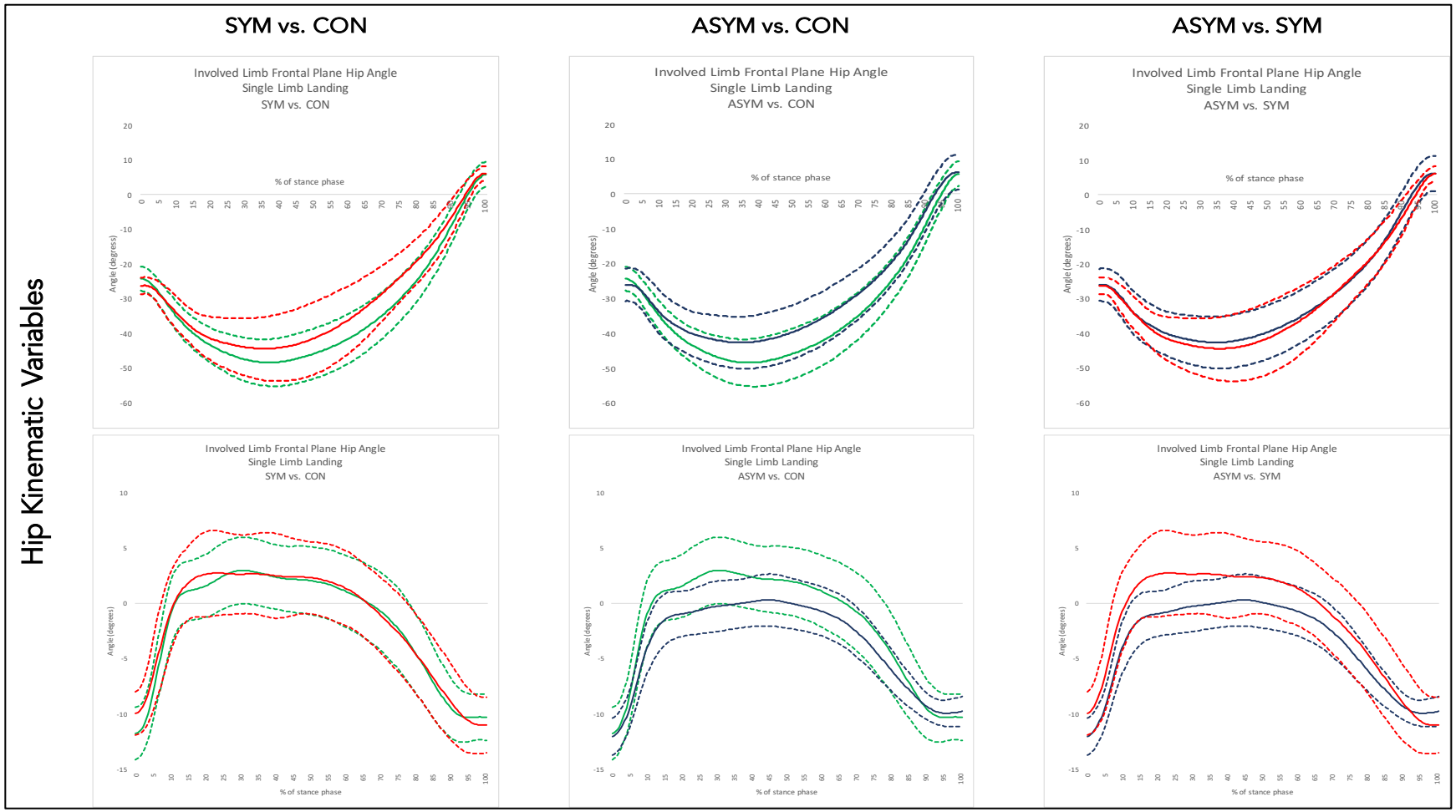


Figure 4.12: Mean and 95% confidence interval waveforms for involved limb knee moment variables during the single-limb jump landing task

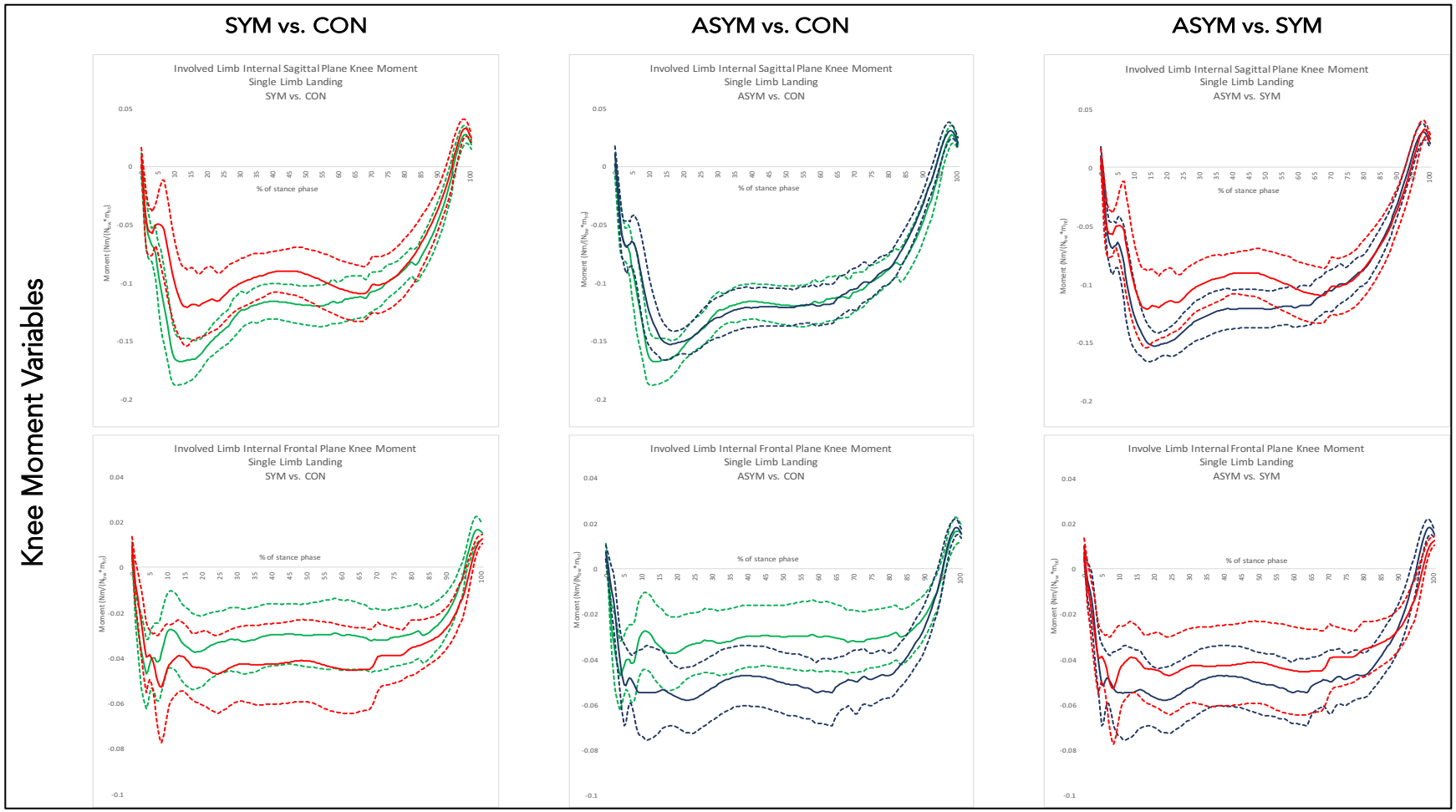


Figure 4.13: Mean and 95% confidence interval waveforms for involved limb hip moment variables during the single-limb jump landing task

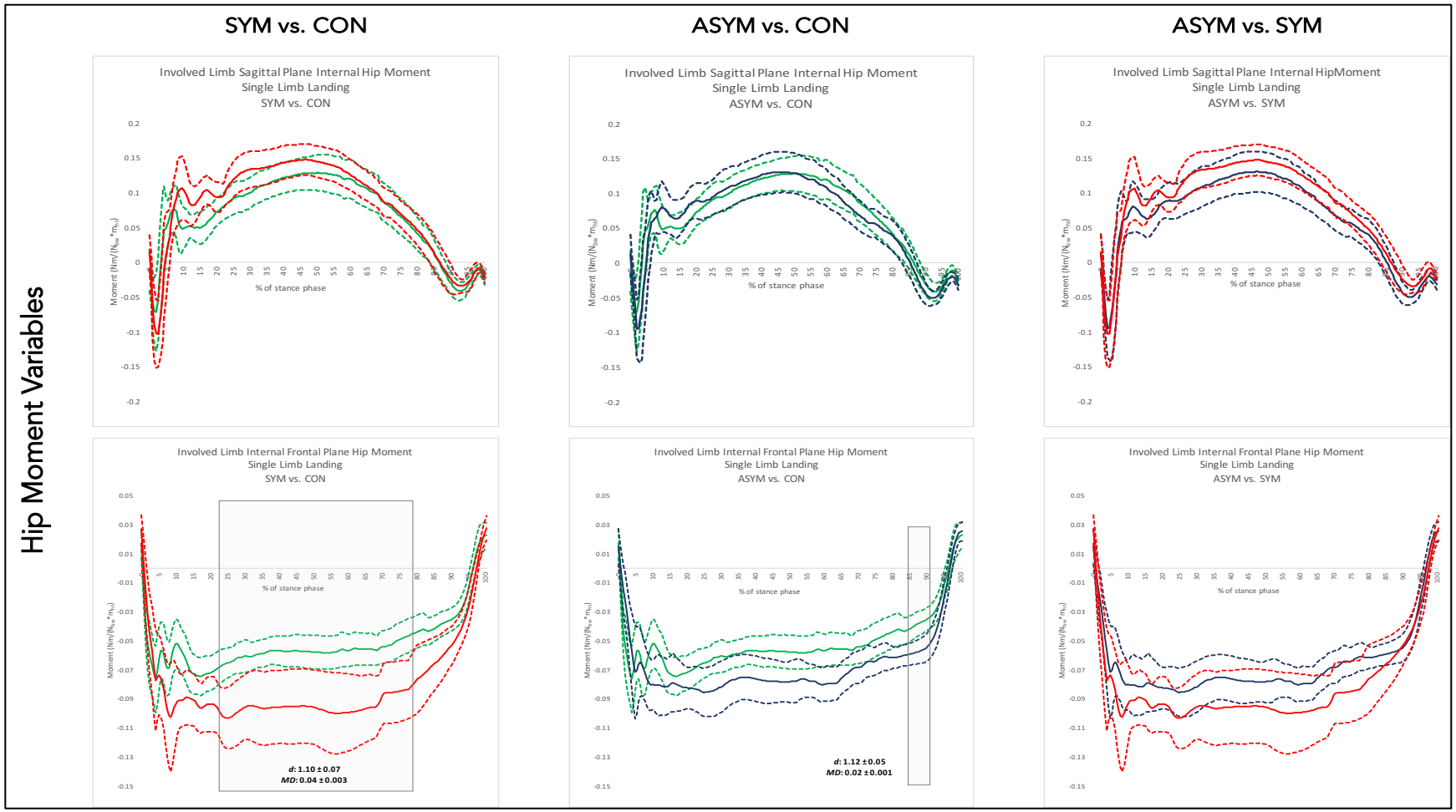


Figure 4.14: Mean and 95% confidence interval waveforms for involved limb vertical ground reaction force (VGRF) and patellar tendon force (PTF) variables during the single-limb jump landing task

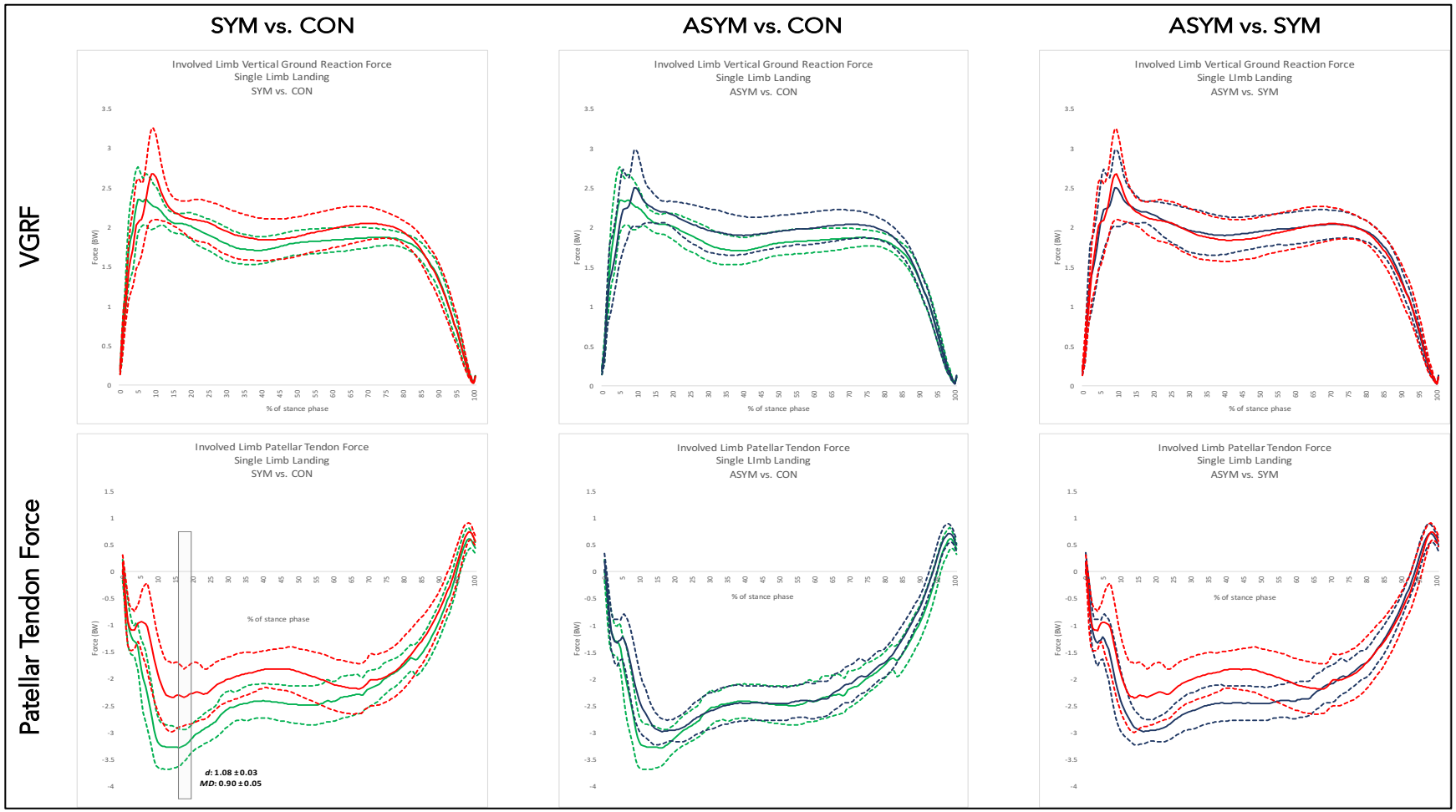
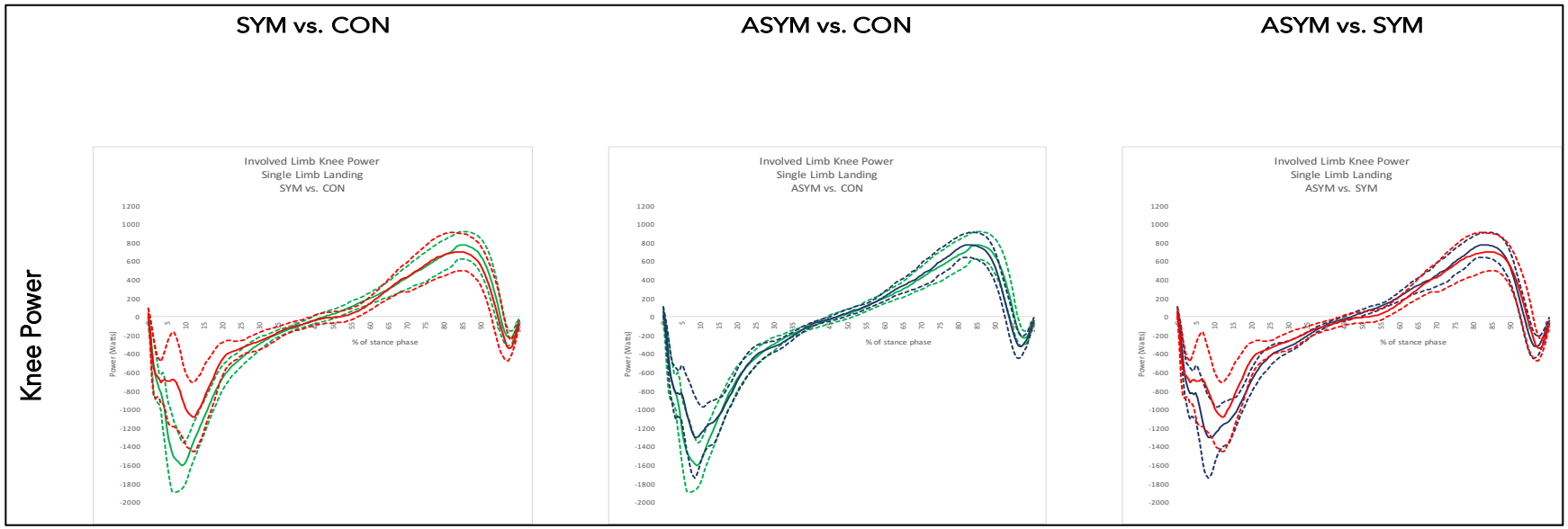


Figure 4.15: Mean and 95% confidence interval waveforms for involved limb knee power variables during the single-limb jump landing task



Specific Aim 2

To ascertain the impact of symptomatic PTA and asymptomatic PTA on cumulative external load during a one-week monitoring period.

Results

No significant differences in height, mass, or age were observed between groups (p > 0.05). The VISA-P score was significantly lower in the SYM-PTA group compared to both the ASYM-PTA and CON groups (p<0.001), and the mean difference exceeded the MCID (13 points) for this subjective outcome measure.[204]

There were no significant differences between the three groups for the load frequency (steps/day, $steps_{MVPA}$/day) or duration (MVPA/day) metrics (p>0.05). However, there was a non-significant trend of fewer steps/day, $steps_{MVPA}$/day, and MVPA/day in the SYM-PTA group compared to both the CON and ASYM-PTA groups. Total wear time was not significantly different between groups (p=0.205). All group comparisons were conducted with and without controlling for total wear time, and neither model demonstrated statistical significance (p>0.05).

Double-Limb Landing

Participants with SYM-PTA demonstrated load volume than CON for all variables (cF_{PT}, $cF_{PT}I$, cKEMI, cKP, cKW) (p<0.05). There were no significant differences between load volume in the SYM-PTA and ASYM-PTA groups or the ASYM-PTA and CON groups (p>0.05). Mean differences and Cohen's d effect sizes are presented in Table 4. The magnitude of the effect for SYM-PTA compared to CON was considered to be strong and significant for cF_{PT} (d =0.98), $F_{PT}I$ (d = 1.09), KEMI (d = 1.09), cKP (d = 10.4), and KW (d = 1.14).

The SYM-PTA group demonstrated significantly less load magnitude than the CON (p<0.01) and ASYM-PTA (p>0.05) groups for all variables ($F_{PT}I$, KEMI, KW). There were no

statistically significant differences in these baseline biomechanical load magnitude variables between the CON and ASYM-PTA groups (p>0.05). The magnitude of the effect for SYM-PTA compared to CON was considered to be strong and significant for $F_{PT}I$ (d = 1.40), KEMI (d = 1.42), and KW (d = 1.53).

Single Limb Landing

The SYM-PTA group demonstrated significantly less load magnitude than the CON (p<0.01) group for all variables ($F_{PT}I$, KEMI, KW). There were no statistically significant differences in these baseline biomechanical load magnitude variables between the CON and ASYM-PTA groups (p>0.05). Mean differences and Cohen's d effect sizes are presented in Table 6. The magnitude of the effect for SYM-PTA compared to CON was considered to be strong and significant for $F_{PT}I$ (d = 1.17), KEMI (d = 1.18), and KW (d = 1.42).

Summary

In summary, male athletes with and without patellar tendinopathy did not demonstrate difference in loading frequency and duration (steps/day and MVPA/day) during a one-week load-monitoring period. However, due to lesser involved limb biomechanical load magnitude during laboratory-based assessment of landing, the SYM group demonstrated significant less cumulative loading volume than the CON and ASYM groups. There were no signs of over-loading in the ASYM group compared to CON group for any load metrics (magnitude, frequency, duration, volume).

The clinical relevance of these findings is that they demonstrate that reductions in load magnitude during an isolated task (i.e. landing) are magnified when extrapolated over longer

periods of time and real-world activity. These findings highlight the importance of a comprehensive approach to load monitoring in individuals with patellar tendinopathy, including biomechanical movement profiles, loading volume, and patient-reported outcomes.

Tables

Table 4.1: Descriptive characteristics of the study population (mean ± sd)

	Healthy Control (n=14)	Asymptomatic Tendinopathy (n=14)	Symptomatic Tendinopathy (n=13)
Age (yrs)	19.64 ± 1.60	21.00 ± 1.96	19.62 ± 1.61
Height (m)	1.84 ± 0.09	1.84 ± 0.07	1.82 ± 0.05
Mass (kg)	79.91 ± 12.95	81.63 ± 13.03	83.46 ± 5.12
Tegner Activity Scale (0-10)	8.00 ± 0.88	8.00 ± 1.04	8.00 ± 1.00
Pubertal Development Scale (0-12)	11.57 ± 0.65	11.86 ± 0.53	11.39 ±0.87
VISA-P (0-100)	97.64 ± 3.41	94.07 ± 7.85	76.15 ± 13.37*^

*: statistically significant difference than CON group (p<0.001, MD: -21.49 (-29.97, -13.01))
^: statistically significant difference than ASYM group (p<0.001, MD: -17.91 (-26.40, -9.44))

Table 4.2: Descriptive characteristics for study population for load frequency and duration metrics (mean ± sd, 95% CI)

	Healthy Control (n=14)		Asymptomatic Tendinopathy (n=14)		Symptomatic Tendinopathy (n=13)	
	Mean ± SD	*95% CI*	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Total steps/day	11,195 ± 1,803	10,154, 12,237	10,143 ± 2,646	8,615, 11,671	10,250 ± 2942	8,472, 12,028
Steps in MVPA/day	7,028 ± 3,329	5,106, 8,951	5,977 ± 2,723	4,405, 7,549	5,487 ± 1716	4,450, 6,524
Time in MVPA/day (min)	102.42 ± 20.73	90.45, 114.40	97.60 ± 37.67	75.85, 119.36	94.54 ± 29.10	76.96, 112.13
Time in MVPA/day (%)	13.27 ± 3.41	11.30, 15.24	11.80 ± 5.57	8.58, 15.02	12.76 ± 4.43	10.08, 15.44
Total wear time (min)	5,298 ± 968	4,739, 5,859	5,217 ± 881	47,08, 5,725	4,598 ± 1,375	4,700, 5,397
Valid wear days	6.58 ± 0.51	6.28, 6.87	6.43 ± 0.85	5.94, 6.92	5.92 ± 0.31	5.25, 6.60

**Note: the values reported are unadjusted for total wear time. No statistical difference between groups for total wear time (p=0.205)*

**No statistically significant differences between groups, both with and without controlling for total wear time (minutes) (p>0.05).*

Table 4.3: Descriptive characteristics (mean ± sd, 95% CI) for load volume variables (based on # of $steps_{MVPA}$ and involved limb biomechanics during the double-limb landing task).

Table 4.3a:

	Cumulative PTF (BW)		**Cumulative PTF Impulse (BW*ms)**		**Cumulative KEM Impulse (Nm*ms)**	
Group	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Healthy Control (n=14)	$-12.3e^3 \pm 6.17e^3$	$-15.9e^3$, $-8.74e^3$	$-2.27e^6 \pm 1.24\ e^6$	$-2.99e^6$, $-1.56\ e^6$	$-10.7e^4 \pm 5.96e^4$	$-14.1e^4$, $-7.24e^4$
Asymptomatic Tendinopathy (n=14)	$-9.97e^3 \pm 4.63e^3$	$-12.5e^3$, $-7.45e^3$	$-1.76e^6 \pm 0.880e^6$	$-2.26e^6$, $-1.25e^6$	$-8.15e^4 \pm 4.04e^4$	$-104.9e^4$, $-5.82e^4$
Symptomatic Tendinopathy (n=13)	**$-7.81e^3 \pm 1.94e^3$***	$-8.98e^3$, $-6.64e^3$	**$-1.22e^6 \pm 0.566e^6$***	$-1.56e^6$, $-8.75e^5$	**$-5.70e^4 \pm 2.56e^4$***	$-7.25e^4$, $-4.15e^4$

Table 4.3b:

	Cumulative Knee Power (J*ms)		**Cumulative Knee Work (J)**	
Group	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Healthy Control (n=14)	$-6.71e^3 \pm 3.46e^3$	$-8.71e^3$, $-4.72e^3$	$-5.56e^5 \pm 3.38e^5$	$-7.51e^5$, $-3.60e^5$
Asymptomatic Tendinopathy (n=14)	$-5.31e^3 \pm 2.59e^3$	$-6.80e^3$, $-3.81e^3$	$-3.70e^5 \pm 1.72e^5$	$-4.70e^5$, $-2.71e^5$
Symptomatic Tendinopathy (n=13)	**$-4.03e^3 \pm 1.18e^3$***	$-4.74e^3$, $-3.32e^3$	**$-2.64e^5 \pm 1.25e^5$^**	$-3.39e^5$, $-1.88e^5$

**statistically significant difference than the healthy control group (p<0.05)*
^ statistically significant difference than the healthy control group (p<0.01)

Table 4.4: Group comparisons for load volume variables (based on # of $steps_{MVPA}$ and involved limb biomechanics during the double-limb landing task).

Table 4.4a:

	Cumulative Patellar Tendon Force (BW)			Cumulative Patellar Tendon Force Impulse (BW*ms)			Cumulative Knee Extension Moment Impulse (Nm*ms)		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	$-4.49e^3$	$-8.76e^3$, $-.21e^3$	0.98	$-10.57e^5$	$-19.44e^5$, $-1.69e^5$	1.09	$-4.99e^4$	$-9.17e^4$, $-.80e^4$	1.09
CON vs. ASYM	$-2.33e^3$	$-6.52e^3$, $1.87e^3$	0.44	$-5.17e^5$	$-13.88e^5$, $3.54e^5$	0.48	$-2.54e^4$	$-6.64e^4$, $1.57e^4$	0.50
ASYM vs. SYM	$-2.16e^3$	$-6.43e^3$, $2.11e^3$	0.64	$-5.40e^5$	$-14.27e^5$, $3.48e^5$	0.73	$-2.45e^4$	$-6.63e^4$, $1.73e^4$	0.72

Table 4.4b:

	Cumulative Knee Power MVPA (J*ms)			Cumulative Knee Work MVPA (J)		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	$-2.68e^3$	$-5.13e^3$, $-.23e^3$	1.04	$-2.92e^5$	$-5.11e^5$. $-.74e^5$	1.14
CON vs. ASYM	$-1.41e^3$	$-3.81e^3$, $1.00e^3$	0.46	$-1.86e^5$	$-4.00e^5$, $.29e^5$	0.69
ASYM vs. SYM	$-1.27e^3$	$-3.73e^3$, $1.18e^3$	0.63	$-1.07e^5$	$-3.26e^5$, $1.12e^5$	0.64

Group Legend: CON=healthy control, ASYM=asymptomatic tendinopathy, SYM=symptomatic tendinopathy
Note: The group listed first is the referent group for each mean comparison.

Table 4.5: Group comparisons for load magnitude variables for the involved (SYM & ASYM) and dominant (CON) limbs during the double limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)		Patellar Tendon Force Impulse (BW*ms)		Negative Knee Work (J/kg)	
	Mean	*95% CI*	*Mean*	*95% CI*	*Mean*	*95% CI*
Healthy Control (n=15)	-29.98 ± 4.91	-32.70, -27.25	-639.20 ± 102.82	-696.14, -582.26	-156.37 ± 34.45	-175.45, -137.29
Asymptomatic Tendinopathy (n=15)	-27.13 ± 5.91	-30.41, -23.85	-583.34 ± 129.80	-655.22, -511.46	-126.58 ± 26.81	-141.43, -111.74
Symptomatic Tendinopathy (n=13)	**-21.19 ± 7.28**$^{*\#}$	-16.79, -25.59	**-450.81 ± 160.57**$^{*\#}$	-547.84, -353.78	**-98.72 ± 40.67**$^{*\#}$	-123.30, -74.15

*statistically significant difference compared to CON group (p<0.01)
#statistically significant difference compared to ASYM group (p<0.05)

Table 4.6: Effect size calculations for group comparisons for load magnitude variables for the involved (SYM & ASYM) and dominant (CON) limbs during the double limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)			**Patellar Tendon Force Impulse (BW*ms)**			**Negative Knee Work (J/kg)**		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	-8.79	-14.37, -3.31	1.42	-188.39	-309.80, -66.97	1.40	-57.65	-89.10, -26.19	1.53
CON vs. ASYM	-2.85	-8.22, 2.53	0.52	-55.86	-172.86, 61.14	0.48	-29.79	-8.22, 2.53	0.97
ASYM vs. SYM	-5.94	-11.52, -0.36	0.90	-132.53	-253.94, -11.12	0.91	-27.86	-11.52, -0.36	0.81

Legend: SYM: symptomatic tendinopathy, ASYM: asymptomatic tendinopathy, CON: healthy control
Note: The group listed first is the referent group for each mean comparison.

The following data outline the findings for load magnitude and load volume variables during the single-limb landing task.

Table 4.7: Comparison of groups for biomechanical energetic variables for involved limb during the single-limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)		**Patellar Tendon Force Impulse (BW*ms)**		**Negative Knee Work (J/kg)**	
	Mean	*95% CI*	*Mean*	*95% CI*	*Mean*	*95% CI*
Healthy Control (n=14)	-40.11 ± 8.89	-45.03, -35.19	-809.80 ± 199.04	-920.03, -699.58	-191,687.81 ± 41,333.18	-214,577.36, -168,798.25
Asymptomatic Tendinopathy (n=14)	-33.94 ± 8.36	-38.57, -29.31	-673.06 ± 170.27	-767.35, -578.77	-155,937.52 ± 50359.60	-183,825.74, -128,049.30
Symptomatic Tendinopathy (n=13)	**-28.72 ± 10.43***	**-35.02, -22.41**	**-569.97 ± 210.86***	**-697.39, -442.56**	**-122,342.77 ± 55151.88***	**-155,670.71, -890,14.82**

*statistically significant difference compared to CON group (p<0.01)

Table 4.8: Effect size calculations for group comparisons for the involved (SYM & ASYM) and dominant (CON) limbs during the single-limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)			**Patellar Tendon Force Impulse (BW*ms)**			**Negative Knee Work (J/kg)**		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	-11.39	-19.89, -2.90	1.18	-239.83	-418.07, -61.59	1.17	-69,345.04	114510.00, -24180.08	1.42
CON vs. ASYM	-6.17	-14.36, 2.01	0.72	-136.74	-308.50, 35.12	0.74	-35750.29	-79272.33, 7771.76	0.78
ASYM vs. SYM	-5.22	-13.72, 3.27	0.55	-103.09	-281.33, 75.16	0.54	-33594.75	-78759.72, 11570.21	0.64

Legend: SYM: symptomatic tendinopathy, ASYM: asymptomatic tendinopathy, CON: healthy control
Note: The group listed first is the referent group for each mean comparison.

The following data outline the baseline limb symmetry indices for each of the landing tasks.

Table 4.9: Single-limb landing task limb symmetry indices (means ± sd and 95% confidence intervals) for the involved (SYM & ASYM) and dominant (CON) limbs

	Internal Knee Extension Moment Impulse (Nm*ms)		**Patellar Tendon Force Impulse (BW*ms)**		**Negative Knee Work (J/kg)**	
Group	*Mean ± sd LSI*	*95% CI*	*Mean LSI*	*95% CI*	*Mean LSI*	*95% CI*
Healthy Control	113.07 ± 29.97	96.48, 129.67	114.39 ± 31.44	96.98, 131.80	115.69 ± 31.35	98.33, 133.05
Asymptomatic Tendinopathy (n	89.60 ± 19.29	78.92, 100.29	89.35 ± 20.28	78.12, 100.58	94.62 ± 32.30	76.73, 112.51
Symptomatic Tendinopathy	107.61 ± 59.56	71.62, 143.60	108.22 ± 62.19	70.63, 145.80	103.28 ± 72.58	59.43, 147.14

Table 4.10: Double-limb landing task limb symmetry indices (means ± sd and 95% confidence intervals) for the involved (SYM & ASYM) and dominant (CON) limbs

	Internal Knee Extension Moment Impulse (Nm*ms)		**Patellar Tendon Force Impulse (BW*ms)**		**Negative Knee Work (J/kg)**	
Group	*Mean ± sd LSI*	*95% CI*	*Mean LSI*	*95% CI*	*Mean LSI*	*95% CI*
Healthy Control	101.00 ±15.42	92.46, 109.54	101.38 ± 15.41	92.85, 109.92	103.37 ± 24.84	89.62, 117.13
Asymptomatic Tendinopathy	97.74 ± 21.45	85.86, 109.62	98.20 ± 22.25	85.88, 110.52	96.45 ± 23.57	83.40, 109.51
Symptomatic Tendinopathy	89.39 ± 31.76	70.20, 108.59	89.21 ± 32.31	69.69, 108.74	92.43 ± 39.99	68.25, 116.60

Legend: SYM: symptomatic tendinopathy, ASYM: asymptomatic tendinopathy, CON: healthy control
*Note: Limb Symmetry Indices (LSI) calculated as: (involved limb/uninvolved limb) * 100, such that values >100 indicate more loading on the involved/dominant limb and values <100 indicate less loading on the involved/dominant limb compared to the referent uninvolved/non-dominant limb, respectively.*
**no significant differences between groups for LSI variables for either landing task (p>0.05)*

Specific Aim 3

To investigate whether an acute isometric patellar tendon loading exercise protocol changes lower extremity landing kinematics and kinetics in individuals with symptomatic and asymptomatic PTA.

Results

No significant differences in height, mass, or age were observed between groups ($p > 0.05$). The VISA-P score was significantly lower in the SYM group than the ASYM group ($p<0.001$), and the mean difference exceeded the MCID (13 points) for this subjective outcome measure (Table 1).[204] There were no group x intervention interactions for change in SLDS NRS pain ($F_{(1, 26)} = 0.555$, p=0.463).

Descriptive characteristics for group and intervention condition are detailed in Table 3. For our within-group comparisons, there was one significant group x intervention interaction for VGRF ($F_{(1, 26)} = 5.33$, $p = 0.029$). However, post-hoc testing with Bonferroni correction ($\alpha = 0.05/4 = 0.0125$) demonstrated no statistical significance. Dependent-samples *t*-tests for each group demonstrated no statistical significance (ASYM: $t = -1.7$, $p = 0.107$; SYM: $t = -1.679$, $p = 0.119$). Independent *t*-tests demonstrated no statistical significance significance (isometric: $t = -2.58$, $p = 0.016$; sham-TENS: 0.72, $p = 0.460$). There were no further significant group x intervention interactions ($p>0.05$). Additionally, the ANCOVA analyses, including baseline biomechanical variables as co-variates, demonstrated the same outcome, as there were no significant group x intervention interactions observed ($p>0.05$).

Summary

We originally hypothesized that a single dose of an isometric loading intervention would result in reduced pain in the SYM group. As a result, we further hypothesized that the SYM

group would demonstrate increased loading on the involved limb during the landing task, including increased FPT impulse, KEM impulse, negative knee work, and knee power. We did not expect to observe any other changes between conditions for either intervention group. In agreement with our hypotheses, there were no changes in biomechanics following the sham-TENS intervention in either group, and there was only one significant change in the ASYM group following the isometric intervention (reduced knee flexion angle at IC). Contrary to our hypothesis the SYM group did not demonstrate significant changes pain or in knee loading biomechanics following the isometric intervention.

The results of this aim demonstrate that an isometric patellar tendon loading exercise protocol did not have acute effects on landing biomechanics in male athletes with symptomatic or asymptomatic tendinopathy. Though isometric tendon loading is a tolerable and analgesic treatment option for athletes with symptomatic patellar tendinopathy,[16–18] our findings suggest that patient selection and duration of intervention implementation may be important factors if using isometric exercise to influence movement profiles. Future research should examine the effects of a longer-duration isometric exercise intervention program on athletes with higher tendon pain and associated disability.

Tables & Figures

Table 4.11: Descriptive characteristics of the study population.

	Asymptomatic Tendinopathy (ASYM) (n=15)	Symptomatic Tendinopathy (SYM) (n=13)
Age (yrs)	21.13 ± 1.88	19.62 ± 1.61
Height (m)	1.84 ± 0.07	1.82 ± 0.05
Mass (kg)	81.45 ± 13.26	83.46 ± 5.12
Tegner Activity Scale (0-10)	7.93 ± 1.03	8.00 ± 1.00
Pubertal Development Scale (0-12)	11.87 ± 0.52	11.39 ±0.87
VISA-P (0-100)	94.40 ± 7.72	76.15 ± 13.37*

*statistically significant difference than ASYM group (p<0.001, MD: -18.25 (-26.41, -10.08))

Table 4.12: Single leg decline squat (SLDS) pain scores (NRS: 0-10) during each testing session.

		Asymptomatic Tendinopathy (ASYM) (n=15)	Symptomatic Tendinopathy (SYM) (n=13)
	Screening Session	0	3.23±1.21
Isometric Intervention Session	Pre-Landing Protocol	0	2.54±1.76
	Pre-Isometric Intervention	0.40±1.55	2.34±2.10
	Post-Isometric Intervention	0.60±1.68	1.62±1.89
	Isometric Intervention Change Score	0.20±0.77	-0.73±0.72
Sham-TENS Intervention Session	Pre-Landing Protocol	0.33±1.29	3.07±1.85
	Pre-Sham TENS Intervention	0.33±1.29	3.03±1.98
	Post-Sham TENS Intervention	0.33±1.29	2.42±1.63
	Sham-TENS Intervention Change Score	0	-0.62±1.12

Table 4.13: Descriptive characteristics (mean difference, standard deviation, 95% CI) for each biomechanical variable change score for the symptomatic and asymptomatic groups for the isometric and sham-TENS intervention conditions.

		Isometric Condition			**Sham-TENS Condition**			*Group x Conditon*	
Biomechanical Variable	**Group**	***Mean* Δ**	***SD***	***95% CI***	***Mean* Δ**	***SD***	***95% CI***	**F**	***p-value***
Knee flexion angle @ IC (°)	SYM	-2.61	3.02	-4.44, -0.78	-1.67	3.15	-3.57, 0.24	1.56	0.223
	ASYM	-4.32	4.45	-6.78, -1.86	-1.57	4.14	-3.87, 0.72		
Peak knee flexion angle (°)	SYM	-3.58	4.91	-6.55, -0.61	-0.79	4.24	-3.35, 1.77	0	0.995
	ASYM	-4.36	7.45	-8.48, -0.23	-1.57	4.72	-4.19, 1.03		
Knee flexion displacement (°)	SYM	-0.97	5.99	-4.59, 2.64	0.88	3.97	-1.52, 3.27	0.44	0.511
	ASYM	-0.04	6.56	-3.67, 3.60	-0.01	4.82	-2.67, 2.65		
Peak VGRF (BW)	SYM	0.46	0.47	0.18, 0.75	0.21	0.23	0.08, 0.35	5.33	**0.029***
	ASYM	-0.04	0.54	-0.34, 0.26	0.32	0.50	0.04, 0.60		
Peak Internal KEM ($N*[kg*m]^{-1}$)	SYM	-0.007	0.26	-0.023, 0.009	0.007	0.03	-0.01, 0.02	2.41	0.132
	ASYM	0.005	0.01	-0.003, 0.01	0.002	0.02	-0.01, 0.02		
Peak F_{PT} (BW)	SYM	-0.14	0.43	-0.40, 0.12	0.20	0.46	-0.08, 0.47	4.06	0.054
	ASYM	0.13	0.32	-0.05, 0.31	0.10	0.38	-0.12, 0.31		
Peak knee power (J/s)	SYM	-0.07	0.38	-0.30, 0.16	-0.03	0.44	-0.29, 0.24	0.21	0.651
	ASYM	0.03	0.28	-0.13, 0.18	-0.02	0.38	-0.24, 0.19		
KEM impulse (Nm*ms)	SYM	-0.44	5.65	-3.85, 2.97	1.63	3.76	-0.64, 3.90	1.86	0.185
	ASYM	2.22	4.56	-0.30, 4.75	1.47	3.93	-0.71, 3.65		
F_{PT} impulse (BW*ms)	SYM	-6.84	123.19	-81.28, 67.60	35.29	80.96	-13.63, 84.21	1.70	0.204
	ASYM	51.13	102.60	-5.68, 107.95	33.94	87.62	-14.58, 82.47		
Negative knee work (J/kg)	SYM	-2.75	26.54	-18.78, 13.29	8.69	25.02	-6.43, 23.81	2.89	0.101
	ASYM	11.71	21.50	-0.20, 23.61	3.85	19.99	-7.22, 14.92		

Legend: **Δ: change**; IC: initial contact; VGRF: vertical ground reaction force; KEM: knee extension moment; F_{PT}: patellar tendon force

*statistically significant at $p < 0.05$.

Table 4.14: Cohen's *d* effect sizes for mean differences (pre-post) within each group for the isometric and sham-TENS intervention conditions.

		Isometric Condition	**Sham-TENS Condition**
Biomechanical Variable	**Group**	Cohen's *d*	Cohen's *d*
Knee flexion angle @ IC (°)	SYM	-0.37	-0.22
	ASYM	-0.92	-0.39
Peak knee flexion angle (°)	SYM	-0.30	-0.06
	ASYM	-0.38	-0.10
Knee flexion displacement (°)	SYM	-0.08	0.07
	ASYM	0.003	0.08
Peak VGRF (BW)	SYM	0.67	0.29
	ASYM	-0.04	0.50
Peak Internal KEM (N*[kg*m]$^{-1}$)	SYM	-0.25	0.24
	ASYM	0.18	0.09
Peak F_{PT} (BW)	SYM	-0.27	0.33
	ASYM	0.27	0.21
Peak knee power (J/s)	SYM	-0.14	-0.05
	ASYM	0.05	-0.03
KEM impulse (Nm*ms)	SYM	-0.06	0.23
	ASYM	0.37	0.18
F_{PT} impulse (BW*ms)	SYM	-0.05	0.23
	ASYM	0.39	0.19
Negative knee work (J/kg)	SYM	-0.07	0.22
	ASYM	0.40	0.09

Figure 4.16: Study CONSORT Diagram

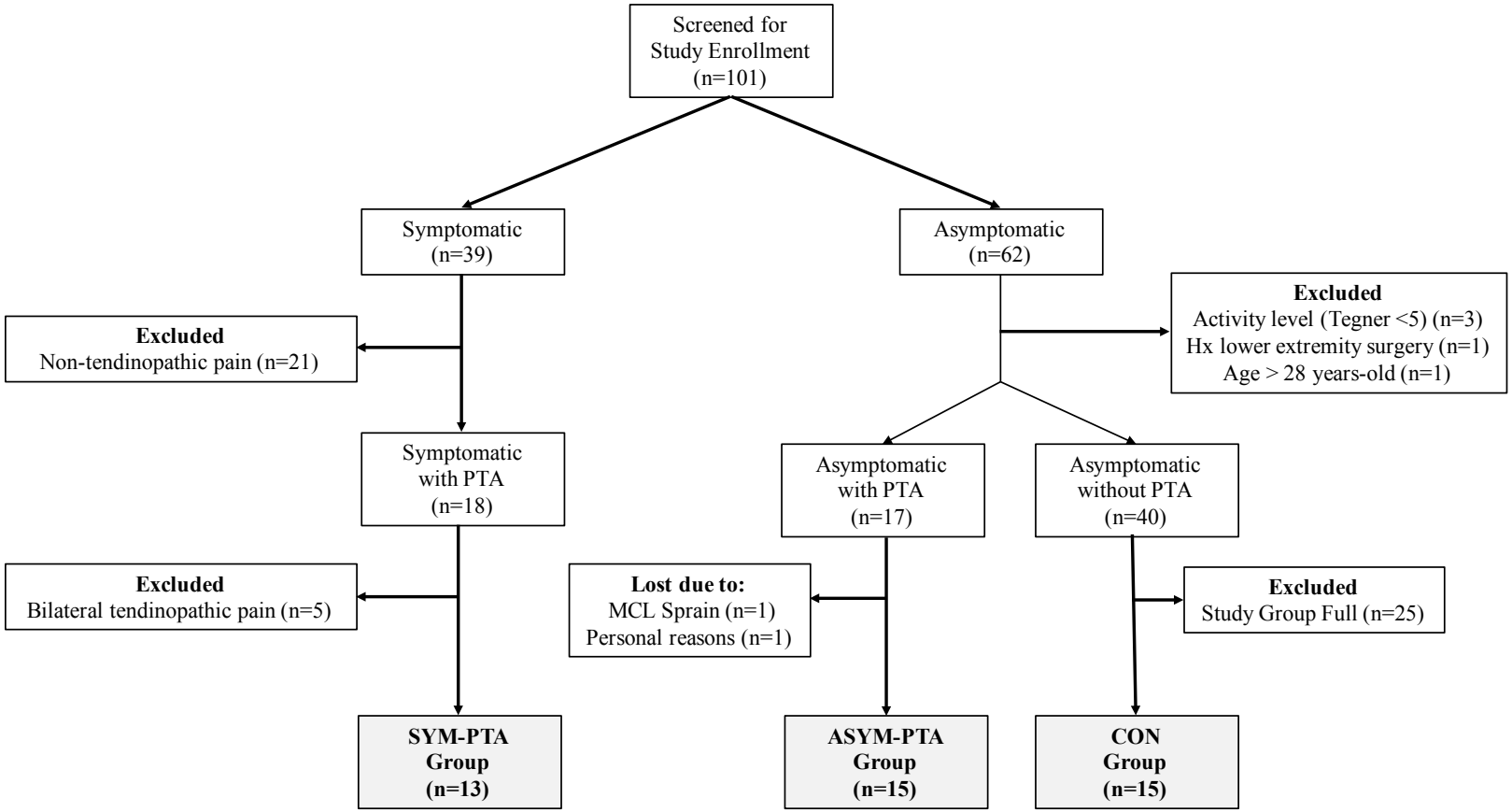


Figure 4.17: Individual participant SLDS pain (NRS: 0-10) change scores following the isometric (blue open circles) and sham-TENS (open red circles) interventions with median group change (black horizontal line).

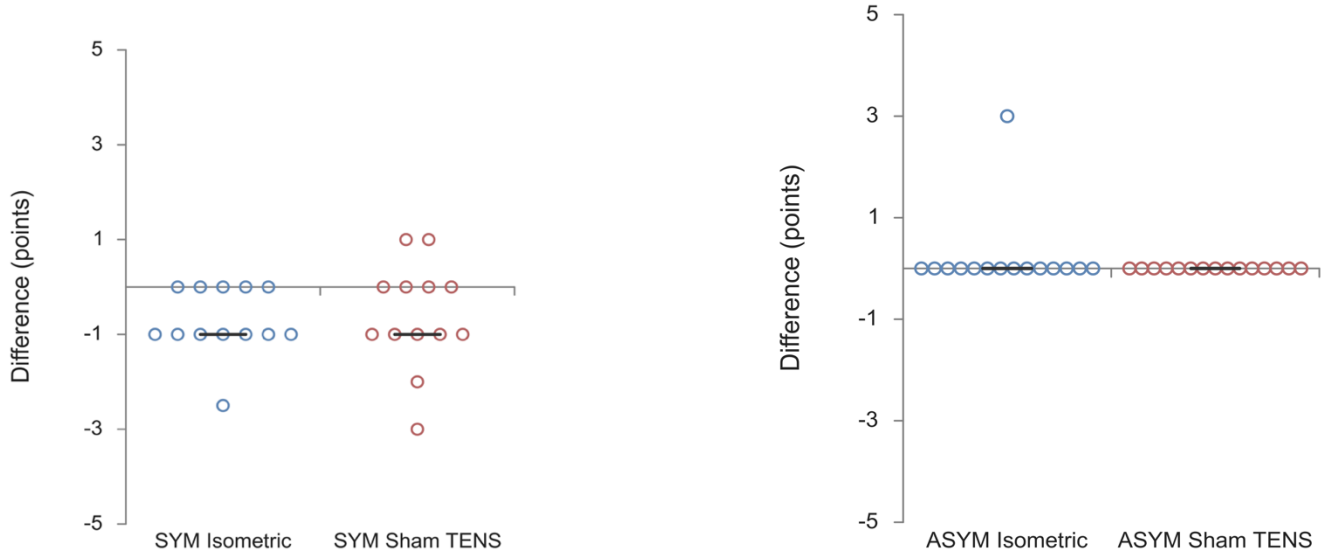


Figure 4.18: Mean and 95% confidence intervals for change scores for isometric and sham-TENS conditions for the SYM and ASYM groups.

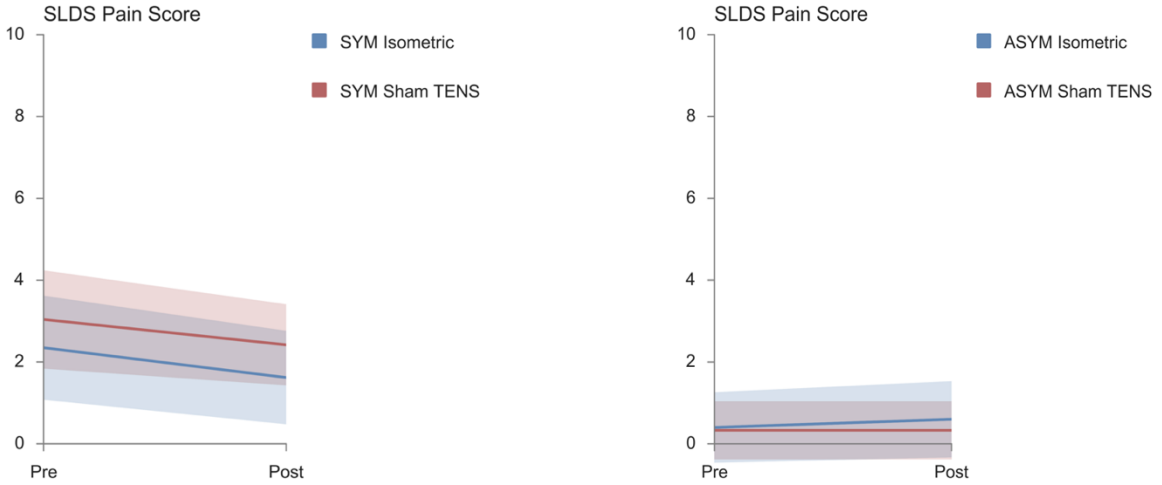
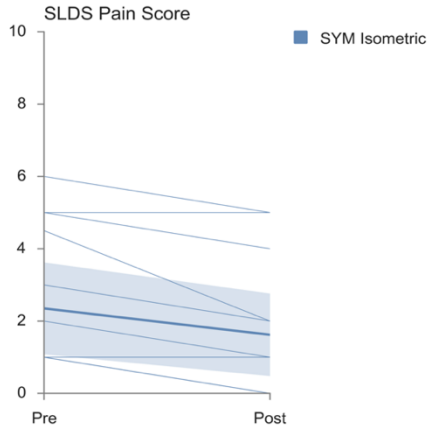
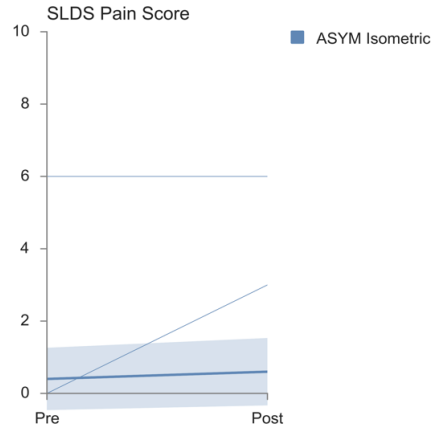


Figure 4.19: Individual participant pre- and post-isometric intervention SLDS pain scores (NRS 0-10) with mean (dark blue line) and 95% confidence bounds (shaded area)

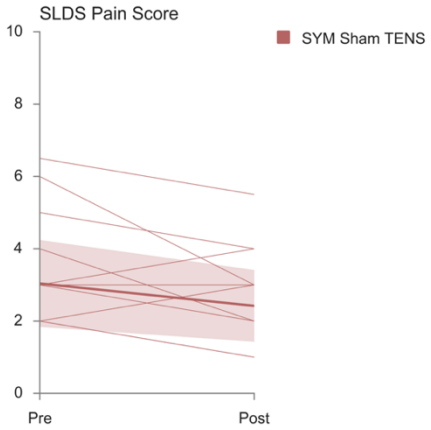


*Note: 2 participants reported pre- and post-NRS=0
2 participants reported pre- and post-NRS=1
3 participants reported pre-NRS=1 and post-NRS=0

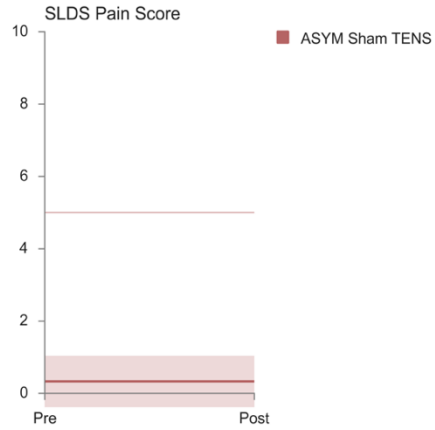


*Note: 13 participants reported pre- and post-NRS=0

Figure 4.20: Individual participant pre- and post-sham-TENS intervention SLDS pain scores (VAS 0-10) with mean (dark red line) and 95% confidence bounds (shaded area)



*Note: 2 participants reported pre-NRS=2 post-NRS=1
1 participant reported pre- and post-NRS=3
1 participant reported pre- and post-NRS =2



*Note: 14 participants reported pre- and post-NRS=0

CHAPTER 5: MANUSCRIPT 1

Differences in Biomechanical Loading Magnitude During a Landing Task in Male Athletes with and without Patellar Tendinopathy

INTRODUCTION

Patellar tendinopathy (PT) is prevalent in individuals who are physically active, particularly athletes who participate in sports with repetitive jumping manueveurs.[1–5,230] While some athletes are able to maintain sport participation, the long-term consequences of chronic tendinopathy include reduced physical activity and quality of life.[6,7] Previous studies have demonstrated differences in lower extremity kinematics, kinetics, and energetics between individuals with and without patellar tendinopathy symptoms,[19–22] as well as between asymptomatic individuals with and without patellar tendon structural abnormalities (PTA).[23,24] However, to-date, there are no biomechanical studies that have compared movement profile characteristics while controlling for both PTA symptoms and simultaneously including a healthy control group (no PTA or symptoms) using robust inclusion criteria. Determining if biomechanical profiles are different between individuals at differing stages along the continuum of tendon pathology[9] may inform the development of enhanced impairment-based, individualized treatment programs.

Individuals with symptomatic patellar tendinopathy tend to employ a tendon-load avoidance movement profile, described as reduced sagittal plane excursion, internal extension moment, and mechanical energy absorption at the knee.[19–22] Rosen et al. (2015) found that symptomatic participants, compared to asymptomatic participants, landed with approximately

degrees lesser peak knee joint flexion and had lesser sagittal plane knee joint excursion during the stance phase of a double-limb jump-landing task.[21] At increasing knee flexion angles, patellar tendon tissue strain increases,[145] so reducing sagittal plane motion during landing supports the expected notion that individuals avoid loading painful tissues during high-energy movements. Furthermore, in a separate study, adult male volleyball athletes with symptomatic PT demonstrated a landing technique that avoids high patellar tendon loads on the involved limb with lesser internal knee extension moment and reduced knee flexion velocity, compared to both those with a previous (>5 months of testing) history of patellar tendinopathy and healthy control participants.[22] Additionally, Sorenson et al. (2010) found that individuals with symptomatic PT had approximately a 30% reduction in net joint work and power and approximately 22% lower peak vGRF than healthy control participants, suggesting reduced mechanical energy absorption and force attenuation on the symptomatic limb.[19]

Though the ideal clinical treatment pathways for asymptomatic individuals with PTA is yet unclear, the presence of a PTA in asymptomatic athletes has been demonstrated as a risk factor for symptom development.[41,43,85,231] Several studies have evaluated biomechanics in asymptomatic individuals with PTA, suggesting a tendon overloading movement profile. In a recent systematic review, van de Worp et al. (2014) found that the greatest differences in kinematic and kinetic variables during landing tasks were present in studies that compared healthy controls to asymptomatic individuals with patellar tendon structural abnormality (PTA).[93] In a comparative cohort of junior pre-elite male basketball athletes with and without PTA, several kinematic variables were found to be predictive of the presence of a PTA, including hip joint excursion and knee flexion angle at initial ground contact. Individuals with a PTA extended their hip joints when landing and had greater knee flexion angles at initial ground

contact.[24] Combined with greater hip flexion angle at initial ground contact,[23,24] this movement strategy is thought to increase the demand on the knee extensor mechanism to counteract the more posterior position of the body's center of mass when transitioning from the loading to propulsive phases of a jump. Furthermore, Edwards et al. (2010) observed individuals with asymptomatic PTA land with more knee flexion at initial contact, go through lesser sagittal plane knee flexion excursion, and demonstrate aberrant hip-knee sequencing patterns, compared to healthy individuals with no symptoms or PTA.[23] This stiff movement profile is thought to reduce inter-joint force distribution and increase combined tensile and compressive strain across the proximal patellar tendon, which has been described as a key mechanical factor leading to tendinopathy.[141]

There are several unique features to the current study that seek to add to the current body of literature around biomechanics and patellar tendinopathy. While previous studies have provided important insights into movement profiles of individuals at various points along the continuum of tendinopathy, it appears that symptomatic and asymptomatic athletes with PTA perform landing tasks with two different movement profiles: under-loading and overloading, respectively. Therefore, studying the role of biomechanics in patellar tendinopathy warrants examination of both distinct groups compared to healthy individuals. Additionally, previous studies use a myriad of diagnostic criteria to define patellar tendinopathy, from self-reported questionnaires and reports of pain during sport,[21,232,233] pain to palpation,[67,136,234] and/or ultrasonographic evidence of structural abnormalities,[2] which makes comparisons across groups and studies challenging. The current study uses an evidence-based battery of tests, including a provocative load-based test for symptoms[110] and diagnostic imaging for PTA,[43,80] to classify stages of PT. Finally, no studies to-date have evaluated movement profiles of individuals with

patellar tendinopathy across the entire stance phase of a landing task, but instead looking at discrete kinematic and kinetic values (i.e. at initial ground contact or at peak), limiting the ability to detect potential differences throughout the remainder of the landing task.

Therefore, the purpose of this study is to compare involved limb biomechanical profiles across the stance phase of a double-limb landing task in three distinct groups: individuals who are symptomatic with PTA, asymptomatic with PTA, and healthy individuals

METHODS

Participants

Forty-three male participants with and without patellar tendinopathy (PT) were enrolled into this study (Table 1). Participants were recruited from the local high school and university communities using approved email correspondence and public flyers. All participants were 15-28 years-old and were required to be actively participating within an organized sport setting, quantified by a Tegner Activity Scale of ≥ 5, a self-reported questionnaire in which the participant identifies the highest level of competition and/or physical activity currently performing.[235] Additionally, all participants were considered to be post-pubertal, quantified by Pubertal Development Scale Stage 5 (score >12).[106,202] This study was approved by the university's institutional review board and informed consent was obtained by all participants prior to study screening.

Screening Protocol for Patellar Tendinopathy

To determine group assignment, all participants underwent a two-part screening protocol. Participants were recruited into the symptomatic PT group (SYM-PTA) if they exhibited: 1) pain ≥2/10 on the numeric rating scale (NRS) only in the patellar tendon during performance of the Single Leg Decline Squat (SLDS) test[110], as selected from a pain map diagram providing a series

of pictures with various anterior knee pain locations (if bilateral pain, the "worse" limb must have been ≥ 5/10 and the contralateral limb must have been ≤ 2/10 on NRS (0-10)), and 2) ultrasonographic (US) evidence of a structural proximal patellar tendon abnormality (PTA), as defined by the presence of a hypoechoic region (≥ 2mm) and/or a maximum thickness of > 7mm, evident on both the longitudinal and transverse scans.[80,85] All US images were obtained and processed by a single trained investigator (L.S.P.). Participants were recruited into the asymptomatic PT group (ASYM-PTA) if they were free of SLDS pain but demonstrated US evidence of a PTA. Finally, participants were recruited into the healthy control group (CON) if free of SLDS pain and PTA.

Any participants were excluded if they exhibited any of the following: 1) known neurological disorders or cardiopulmonary diseases, 2) a history of any lower extremity surgery, 3) a history of a lower extremity injury in the prior six-months, 4) an injection to the patellar tendon in the prior last three-months, 5) participation in formal rehabilitation for anterior knee pain in the prior three-months, 6) presentation of non-tendinopathic knee pain during the SLDS test (i.e. patellofemoral pain syndrome presentation), or 7) any other medical condition that would prevent them from participation in normal activities of daily living.

Patient-Reported Outcomes

The Victorian Institute of Sport Assessment-Patellar Tendon (VISA-P) questionnaire was used to quantify self-reported knee function.[203,204,236] The VISA-P consists of 8-items regarding the presence of pain during various daily and sport-related activities and has demonstrated excellent test-retest and interrater reliability.[236] All participants completed this questionnaire at the time of the screening session.

Three-Dimensional Landing Assessment

Testing Protocol

On a single testing day, participants visited the laboratory for a three-dimensional biomechanical landing assessment. Participants performed a 5-minute warm-up on a stationary bicycle at a self-selected pace.

Double-Limb Jump Landing Task

Participants were provided with spandex shorts and tops and wore their own athletic shoes. Participants performed five trials of a jump-landing task from a 30 cm box that was positioned 50% of the participant's height from the front edge of the force plates.[221] The participants were instructed to jump forward off the box to a double-leg landing with one foot on each force plate, and immediately perform a maximal vertical jump upon landing.[221] A minimum of one practice trial was performed; practice trials were performed until the participant and investigator ensured correct performance of the jump-landing task. A total of five jump-landing trials were collected, and the middle three trials were averaged for data analysis. If one of the middle three trials was not successful, a subsequent trial was utilized for analysis. A successful trial required the participant to leave the box with both feet at the same time, land on the force plates, and jump straight up in the air as high as possible.

Participants were outfitted with 20 retro-reflective markers bilaterally on the following bony landmarks: acromion process, anterior superior iliac spine (ASIS), greater trochanter, medial and lateral femoral condyles, medial and lateral malleoli, calcanei, and the first and fifth metatarsal heads.[219] A single marker was placed on the manubrium of the sternum and at the L4-L5 vertebral space. Rigid clusters of three or four markers was placed at the sacrum and on the thigh, shank, and foot segments bilaterally. A static trial was captured with the participant

standing with arms positioned at 90° of shoulder abduction to estimate the location of the landmarks needed to calculate joint centers.[219] After the static trial, the single markers on the foot, malleoli, femoral condyles, and greater trochanters were removed.

Data Acquisition

Three-dimensional kinematic data were collected using a ten-camera motion capture system (Vicon Motion Systems, Centennial, CO, USA) sampled at 120Hz and filtered using a fourth-order low-pass Butterworth filter with a 20Hz cutoff frequency. Kinetic data were sampled at 1200Hz using two floor embedded force plates (Bertec Corporation, Columbus, OH, USA). Knee and ankle joint center coordinates were defined as the centroid between the medial and lateral condyles and malleoli identified during the static trail. Hip joint center coordinates were estimated from the coordinates of the L4-5, right ASIS, and left ASIS markers using the Bell method.[222] Reference frames for the foot, tibia, and femur were defined based on 3D-coordinates and segments as follows: 1st and 5th metatarsal heads, ankle joint center, and calcaneus (foot); medial and lateral malleoli, knee and ankle joint centers, and shank (tibia); medial and lateral femoral condyles, knee and hip joint centers, and thigh (femur). Joint angles were defined based on the position of the distal segment relative to the proximal segment using a Cardan angle sequence in the following order of rotation: sagittal (y-axis), frontal (x-axis'), and transverse (z-axis').

Data Processing & Reduction

Marker coordinate and ground reaction force data was transferred into The Motion Monitor software (Innovative Sports Training, Chicago, Illinois) to build three-dimensional link-segment models for biomechanical data analysis and reduction. Lower extremity biomechanics for each limb were evaluated during the stance phase, which was defined as the interval from

initial contact (IC) to toe-off.[223] IC was defined as the time point when the vertical ground reaction force (vGRF) exceeded 10N and toe-off as the time point when vGRF dropped below 10N.[223]

Kinematic variables of interest for this study included knee flexion (+)/extension (-). Kinetic variables of interest included vertical ground reaction force (VGRF), internal knee extension moment (KEM), patellar tendon force (F_{PT}), and knee power. Ground reaction force data and processed segment data were used to calculate net internal sagittal and frontal plane knee joint moments using inverse dynamics procedures.[146] Patellar tendon force (F_{PT}) was estimated using previously defined methodology of Nisell and Ekholm, through which F_{PT} is calculated by dividing the internal knee extension moment by the patellar tendon moment arm.[160] PT moment arms were calculated as a function of knee joint angles using the methodology of Herzog and Read.[144] Internal moments were normalized to the product of the participant's body weight and height ($N*m[kg*m]^{-1}$), while vGRF and F_{PT} was normalized to body weight (BW). Knee power (J/s) was calculated as the product of the internal sagittal plane knee moment ($N*m[kg*m]^{-1}$) * knee flexion velocity (°/millisecond).

Kinematic and kinetic variables were analyzed as continuous normalized waveforms during the stance phase of the landing tasks using custom MATLAB code (MatLab R2017b, The MathWorks, Inc., Natick, MA). For each dependent variable, the within-group mean values were interpolated and normalized over 202 data points over the stance phase using a cubic spline filter.[237] These data points represent 0% - 100% of the stance phase of the landing task (IC through toe-off).

STATISTICAL ANALYSIS

Demographic data was compared across groups using a one-way analysis of variance and Tukey post-hoc tests for pairwise comparisons with SPSS v22 (IBM Inc., Armonk, New York, USA) (Table 1). Kinematic and kinetic group mean values were calculated for each 1% of the landing task, and plotted along with 95% confidence intervals (CI) for each group comparison. Statistical significance was defined as any area of the stance phase where the 95% CI did not overlap for a minimum of a consecutive 3% of the stance phase.[38,237,238] Average mean differences (MD) and Cohen's *d* effect sizes were calculated for any statistically significant areas. Cohen's *d* effect sizes were classified as weak ($d \leq 0.2$), small ($d = 0.2$-0.5), moderate ($d = 0.5$-0.8), or large ($d \geq 0.8$).[226]

Figure 1: CONSORT diagram for study recruitment and enrollment.

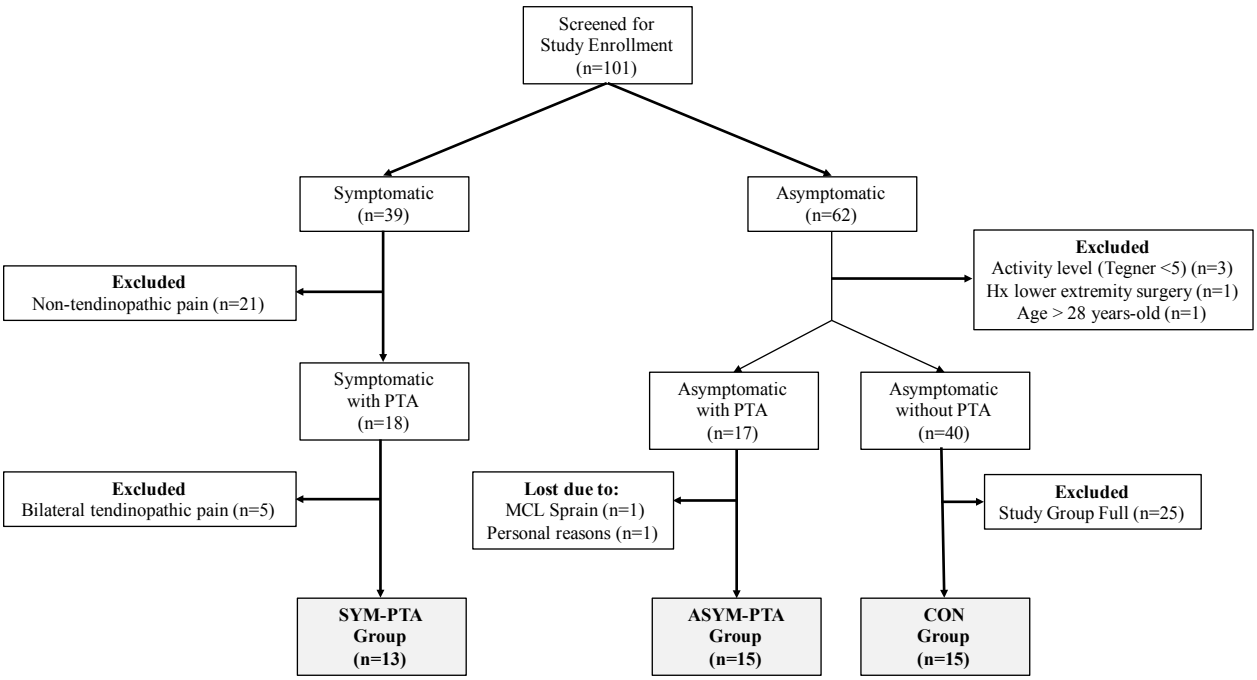


Table 1: Descriptive Characteristics of the Study Population

	Healthy Control (n=15)	Asymptomatic Tendinopathy (n=15)	Symptomatic Tendinopathy (n=13)
Age (yrs)	19.60 ± 1.55	21.13 ± 1.88[#]	19.62 ± 1.61
Height (m)	1.84 ± 0.09	1.84 ± 0.07	1.82 ± 0.05
Mass (kg)	79.08 ± 12.37	81.45 ± 13.26	83.46 ± 5.12
Tegner Activity Scale (0-10)	8.07 ± 0.88	7.93 ± 1.03	8.00 ± 1.00
Pubertal Development Scale (0-12)	11.60 ± 0.63	11.87 ± 0.52	11.39 ±0.87
VISA-P (0-100)	97.80 ± 3.34	94.40 ± 7.72	76.15 ± 13.37[*^]
Screening SLDS Pain (NRS: 0-10)	---	---	3.69 ± 1.25
Pre-Testing SLDS Pain (NRS: 0-10)	---	---	2.38 ± 1.61

[#]: statistically significant difference than CON group (p=0.045)
[*]: statistically significant difference than CON group (p<0.001, MD: -21.65 (-29.81, -13.48))
[^]: statistically significant difference than ASYM group (p<0.001, MD: -18.25 (-26.41, -10.08))

RESULTS

The CONSORT diagram for recruitment and enrollment is detailed in Figure 1. Demographic data are presented in Table 1. No significant differences in height and mass were observed between groups (p > 0.05), but the ASYM group was slightly older than the CON group (p=0.045). The VISA-P score was significantly lower in the SYM group compared to both the ASYM and CON groups (p<0.001), and the mean differences exceeded the MCID (13 points) for this subjective outcome measure.[204] There were no differences in VISA-P score between the ASYM and CON groups (p>0.05).

Kinematics

Participants in the SYM group demonstrated lesser magnitude knee flexion angle than the CON group throughout the majority of the stance phase (8-76%, *d*: 1.14±0.12, Mean Difference (MD): 15.83±2.71°). Participants in the ASYM group demonstrated lesser magnitude knee flexion angle than the CON group during the early (8-13%, *d*: 0.99 ± 0.04, MD: 7.99±0.39°; 21-24%, *d*: 1.01±0.01, MD: 11.11±0.32°) and late (74-94%, *d*: 0.96 ± 0.07, MD: 9.55 ± 1.13°)

portions of the stance phase (Figure 2). There were no differences between the SYM and ASYM groups in sagittal plane knee angle.

Kinetics

Participants in the SYM group demonstrated lesser internal knee extension moment than the CON group during early stance (6.5-9%, *d*: 1.21± 0.08, MD: 0.04 ± 0.004 N*m[kg*m]$^{-1}$), as well as the ASYM group during mid-stance (38-56%, *d*: 1.17 ± 0.06, MD: 0.03 ± 0.001 N*m[kg*m]$^{-1}$) (Figure 4). There were no differences between the ASYM and CON groups in internal sagittal plane knee moment.

There were no differences in vGRF between groups (Figure 3). However, the SYM group demonstrated less patellar tendon force during early stance (6-9%, *d:* 1.15 ± 0.15, MD: 0.85 ± 0.15 (BW)) than the CON group and during mid-stance (36-60%, *d*: 1.22 ± 0.08, MD: 0.66 ± 0.05 (BW)) than ASYM group (Figure 5). There were no differences between the ASYM and CON groups in patellar tendon force. Finally, participants in the SYM group had less knee power during early (6-9%, *d*: 1.24 ± 0.17, MD: 0.48 ± 0.16 J/s; 18.5-23%, *d*: 1.34 ± 0.13, MD: 0.17 ± 0.01 J/s) than the CON group and during early stance (20.5-25%, *d*: 1.14 ± 0.08, MD: 0.20 ± 0.01 J/s) than the ASYM group (Figure 6). There were no differences in knee power between the ASYM and CON groups.

Figure 2: Mean and 95% confidence interval waveforms for involved limb sagittal plane knee motion during the double-limb jump landing task.

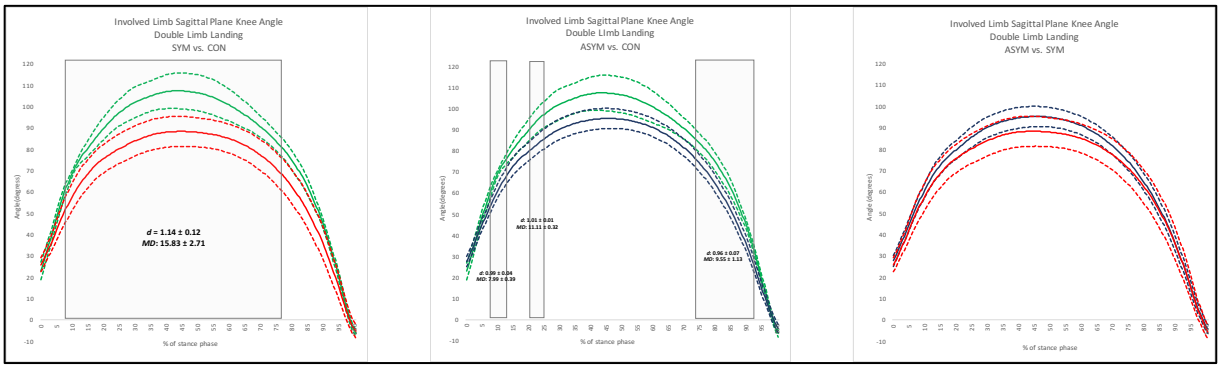


Figure 3: Mean and 95% confidence interval waveforms for involved limb vertical ground reaction force during the double-limb jump landing.

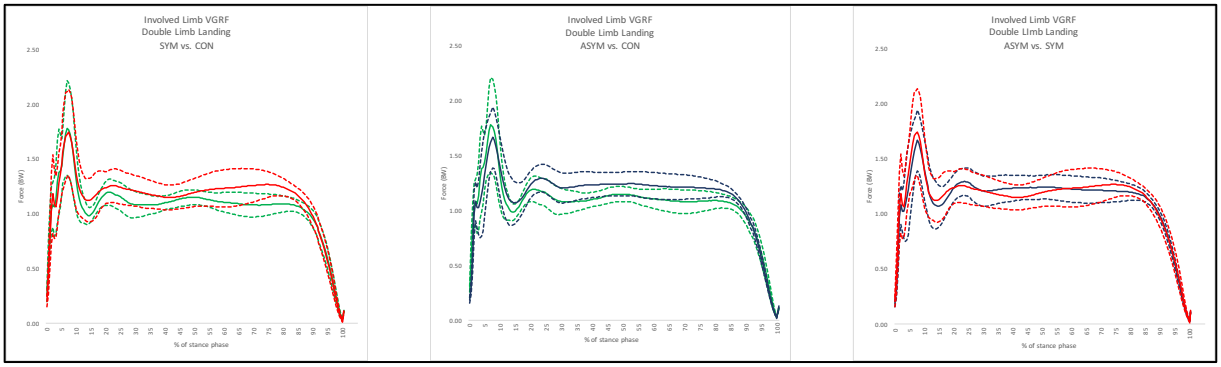


Figure 4: Mean and 95% confidence interval waveforms for involved limb sagittal plane internal knee moment during the double-limb jump landing.

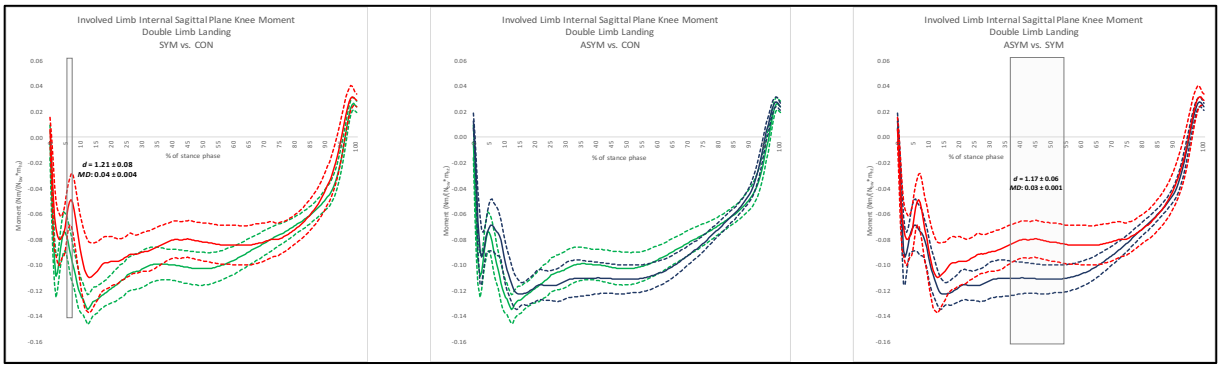


Figure 5: Mean and 95% confidence interval waveforms for involved limb patellar tendon force during the double-limb jump landing.

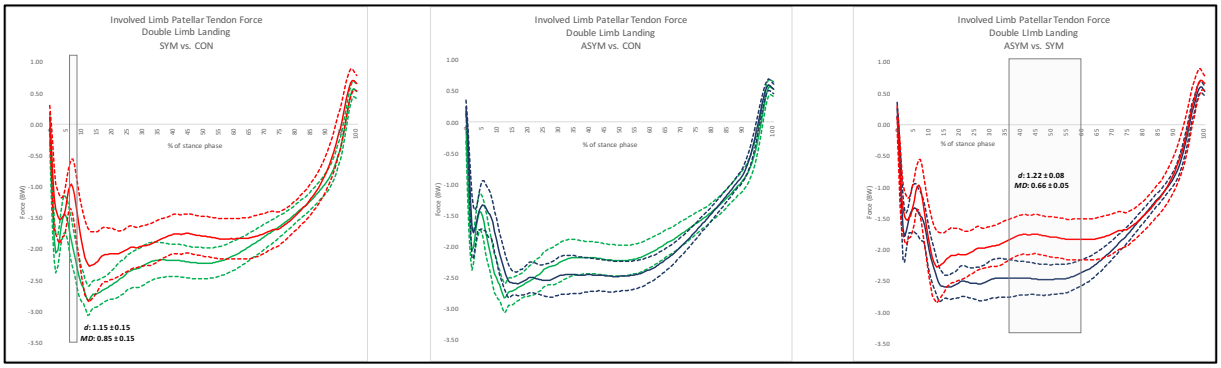
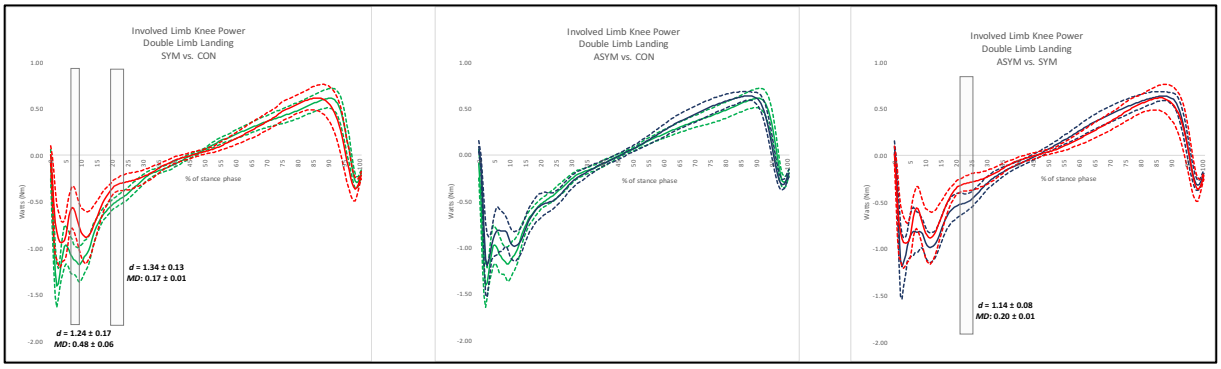


Figure 6: Mean and 95% confidence interval waveforms for involved limb knee power during the double-limb jump landing.



DISCUSSION

The key finding of this study is that male athletes with symptomatic patellar tendinopathy demonstrate a tendon load-avoidance movement profile on the involved limb, demonstrated by reduced sagittal plane knee joint motion, patellar tendon load, and energy absorption at key portions of the stance phase of a double-limb landing task compared to both asymptomatic participants with PTA and healthy controls. A second key finding is that, contrary to our hypothesis, asymptomatic participants with PTA did not demonstrate tendon overloading

movement profiles, suggesting that the increased mechanical loading may not be major factor driving the development of symptomatic tendinopathy. This study is novel in that it is the first to directly compare landing biomechanics in three distinct groups with and without tendinopathy, defined by both structural and load-based symptom assessments. Our findings have important clinical implications to inform rehabilitation programs targeting movement retraining in individuals along the continuum of patellar tendinopathy.

The knee extensor mechanism endures high magnitudes of kinetic energy during landing, with upwards of 7x BW of force placed on the patellar tendon.[23,159] As such, biomechanical analyses that examine tendon load during the entire energy absorption phase of a landing task, such as those used in the current study, may provide a comprehensive understanding of the total mechanical loading demands.

Symptomatic Patellar Tendinopathy Group

The biomechanical profile of the SYM group demonstrates a general pattern of under-loading. Early in the energy absorption phase of the landing task, the SYM group demonstrated lesser internal knee extension moment compared to the CON group (*d*: 1.12, MD: 0.04 ± 0.004 N*m[kg*m]$^{-1}$). In this same early phase of the landing task, we also observed less patellar tendon force F_{PT} in the SYM group compared to CON group (*d*: 1.15, MD: 0.85 ± 0.15 (BW)). Finally, there was also less knee joint power during this landing phase in the SYM group compared to CON group (*d*: 1.24, MD: 0.48 ± 0.06 W(Nm)), indicating a reduction in the rate at which the internal knee extension moment absorbs during the eccentric phase. Paired with reduced sagittal plane motion, this biomechanical profile is suggestive of a quadriceps-avoidance loading pattern, reducing the demand on the extensor mechanism (quadriceps muscle/tendon and patellar tendon) to control the external knee flexion moment.

Our findings of an under-loading biomechanical profile in those with SYM PT is consistent with previous research.[21,239] Reduced peak knee flexion motion and knee flexion excursion during landing tasks have been noted in individuals with current[21] and a history of previous (≥5 months)[239] symptoms compared to healthy controls. Conversely, Richards et al. (1996) observed greater knee flexion motion in male elite volleyball athletes with patellar tendinopathy.[156] However, this is the only study demonstrating elevated sagittal plane kinematics in symptomatic individuals, the results of this study should be interpreted cautiously due to the low small sample of athletes with tendinopathy (n=3), and the exclusive use of palpation as the diagnostic inclusion criteria for tendinopathy.

Importantly, in previous studies, symptomatic PT has been typically defined based on self-reported pain and/or pain with palpation, without confirmation of a PTA. The sensitivity (68%) and specificity (9%) of palpation is poor, and thus palpation is not considered a robust diagnostic tool.[108] Additionally, numerous overuse injury conditions, such as patellofemoral pain syndrome, commonly present clinically with activity-related anterior knee pain. Utilizing a systematic and comprehensive approach to define PT based on both tendon pain and tendon structural abnormality is an important and novel feature of the current study.

Interestingly, no participants in the SYM group reported pain during testing that prevented completion of the jump-landing task, despite the presence of SLDS pain immediately prior to testing. However, they did report significantly more pain during activity and sport than the other groups, as quantified by the low average VISA-P score (76.15 ± 13.37). While we did not account for the duration of activity-related tendon pain, we hypothesize that these simultaneous kinetic and energetic alterations were likely learned behaviors over time to reduce tendon load due to the presence of activity-relate pain. This finding suggests that closely

monitoring activity-related pain and cumulative training load may be an important feature of both prevention and treatment programs for PT, as the high frequency of loading during sport-related activities may be more provocative and more noticeable to a patient than during a single bout of landings, as used in this study.

Asymptomatic Patellar Tendinopathy Group

Unlike previous studies,[23,24] our findings demonstrate that athletes with asymptomatic PTA did not demonstrate a biomechanical profile suggestive of over-loading. In contrast, we observed patterns of under-loading. The ASYM group demonstrated reduced sagittal plane knee motion in the ASYM group compared to CON group during early (8-13% and 21-24%) and late (74-94%) phases of the landing task ranging from 8-11° with large effects (*d*: 0.96-1.01). In addition, there were no differences in peak knee flexion angle, VGRF, KEM, PTF, and knee power between the ASYM and CON groups. Overall these findings indicate that male athletes with asymptomatic PTA did not exhibit any evidence of over-loading across the entire stance phase of the landing task, which may have important clinical implication for the best treatment approach for this patient population.

The lack of evidence of over-loading in those with asymptomatic PTA in the current study is in contrast with prior research. In a previous study, asymptomatic adult male athletes with PTA demonstrated greater sagittal plane knee motion at initial ground contact, but less knee flexion displacement compared to healthy controls during the horizontal phase of a landing task.[23] Additionally, a comparative cohort of junior pre-elite male basketball asymptomatic athletes with and without PTA found that asymptomatic athletes with PTA had greater knee flexion angles at initial ground contact.[24] These movement strategies have been hypothesized to facilitate a stiff landing movement profile that increases the demand on the knee extensor

mechanism, which may increase the combined tensile and compressive strain across the proximal patellar tendon.[141] However, our findings demonstrate that those with asymptomatic PTA display similar VGRF, KEM, PTF, and knee power loading profiles compared to healthy controls and those with symptomatic PTA.

Due to the cross-sectional nature of this study, we are unable to determine whether the observed biomechanical profile of the ASYM group were present prior to the development of the PTA or resulted in response to the PTA. Therefore, the hypothesis that the progression of asymptomatic to symptomatic PTA may be related to increased PT stress from greater amounts of sagittal plane knee motion can neither be supported or refuted from our results. Longitudinal studies are needed to determine whether biomechanical movement profiles influence the initial development of PTA. One such study has been published which found no significant kinematic patterns that differed between athletes who went on to develop patellar tendinopathy.[240] However, of forty-nine participants, only three developed tendinopathy, which was defined based on a clinical diagnosis of pain during loading and palpation without any diagnostic imaging, so results should be interpreted cautiously in comparison with the present study. Interestingly, in the current study, the lesser knee flexion motion in the ASYM vs. CON groups was observed with no simultaneous differences in knee loading variables between these groups. Early alterations in kinematics may proceed changes in loading, particularly during a double-limb landing task where the contralateral limb can be utilized to share force dissipation.

It is possible that, despite the absence of pain in the ASYM group, reduced involved limb sagittal plane motion is a biomechanical adaptation occurring in response to an underlying catabolic biochemical process within the tendon structure. Normal tendon's extracellular matrix is largely composed of tightly packed, highly-organized Type I collagen, the primary load-

bearing structure of tendon, and is supported by tenocytes and small proteoglycans.[241,242] Mechanoreceptors in tendon are located primarily at myotendinous junctions and tendon insertions. The cell proliferation and matrix disorganization in tendon pathology includes the production of larger proteoglycans, which alters mechanotransduction and disrupts tendon molecular homeostasis.[243,244] It is hypothesized that the asymptomatic tendon may have considerable matrix disorganization and disruption of normal mechano-transductive properties, but inadequate chemical nociceptive stimulus to cause perceived pain.[82]

Therefore, we hypothesize that the reduction in knee flexion motion in asymptomatic participants compared to healthy controls may be an underlying avoidance of a joint position that places high demand on the abnormal tendon that possesses compromised mechano-transductive capabilities. It is also possible that these individuals have a different inherent tissue capacity such that even though they experience similar load magnitudes as healthy individuals, their ability to withstand those loads without tissue compromise is reduced. Finally, it is possible that loading characteristics measured in controlled laboratory settings are failing to tell the entire story of the cumulative effects of loading on the development of PT, warranting further investigations into how loading in real-world athletic settings may influence the development and progression of patellar tendinopathy.

We believe that our findings are important, as they suggest that interventions for asymptomatic individuals with structural abnormalities should not be designed to shield the tendon from excessive loading, as these individuals are already demonstrating signals of under-loading. Our data show that these asymptomatic individuals land with reduced knee flexion and, though not statistically significant, are trending towards reduced knee kinetics compared to healthy controls.

This movement pattern may lead to reduced tissue capacity and subsequent symptom development over time. Recent work by Docking and Cook (2015) found that pathological tendons have increased cross sectional area of aligned fibrillar structure around areas of pathology, suggestive that tendon may actually adapt to pathology.[80] This thickening of healthy tendon results in sufficient amounts of load-bearing tissue that should be targeted through progressive load-based interventions to build overall tissue capacity. While there is evidence suggesting that degenerative tendon matrix is unlikely to be reversible, this evolving concept focused on "treating the doughnut (aligned structure), not the hole (area of disorganization)".[10] is potentially an area that can improve the function and prognosis of asymptomatic individuals with structural abnormalities.

LIMITATIONS

We acknowledge several limitations to this study. The cross-sectional design of this study prevents us from determining whether the observed movement profiles were present prior to the development of PTA with or without symptoms in our SYM and ASYM groups, respectively. While robust inclusion criteria were utilized to define our tendinopathy groups, we did not account for duration of symptoms in the SYM group, which may have influenced how long an individual may have developed and instilled aberrant movement profiles in response to persistent pain. In addition, our SYM cohort was relatively functional, based on low to moderate pain levels reported prior to the biomechanical assessment. We also recognize that more between-group differences in movement profiles may have been absent due to the relatively sample size for each group, as well as the high degree of variability, particularly in the SYM group, whose 95% CI were wide. While our estimation of patellar tendon force was chosen based on accepted models,[23,144,158,160] it is possible that these models underestimate the actual force acting through

tendon. Finally, this study was conducted only on college-aged males so its findings cannot be extrapolated to females or males of a different age range.

CONCLUSIONS

Several important inferences can be made from the results of this study that carry meaningful clinical implications. Participants with symptomatic tendinopathy demonstrated differences in both sagittal plane kinematic and kinetic movement profiles during portions of the stance phase of a landing that are associated with high patellar tendon stress and extensor mechanism demand, respectively. This study is the first to use magnitude-based inference waveform analysis to evaluate landing characteristics in patellar tendinopathy. We believe this type of analysis may be more effective at comprehensively evaluating movement characteristics, as we were able to highlight group differences in phases of the tasks that would not have been exposed using traditional discrete variable analysis. Second, though the periods of reduced loading in the symptomatic group were relatively short, if uncorrected over time, an altered movement strategy of under-loading may lead to both reduced tissue capacity and reduced performance. Future studies should continue investigating the best treatment options to reduce pain and re-train normal tendon loading to maximize the quality of load-bearing capabilities of tendon.[188] Finally, reductions in sagittal plane knee motion of the asymptomatic participants compared to the healthy control participants may indicate a compensatory strategy in response to underlying tissue pathology. Consequently, regular observation of individuals with structural abnormalities who are at higher risk for developing symptoms is warranted. Future research should continue investigating both laboratory and real-world movement characteristics of individuals at varying stages of the continuum of tendinopathy in order to best design targeted rehabilitation strategies to improve tissue resilience and performance.

CHAPTER 6: MANUSCRIPT 2

Load Magnitude and Cumulative Load Volume Differ in Male Athletes with and without Patellar Tendinopathy

INTRODUCTION

Patellar tendinopathy (PT) is prevalent in individuals who are highly physically active, particularly athletes who participate in sports with repetitive jumping manueveurs.[1–5] Mismanagement of the volume of load acting on the tissue is believed to be a key factor contributing to PT development.[9,10] Research involving biomechanical analyses has revealed differences in the load magnitude of vertical ground reaction force (VGRF), knee extension moment (KEM), and patellar tendon force (F_{PT}) between individuals with and without symptoms of PT.[19,22,23] A limitation of traditional biomechanics research is that it provides a snapshot of the individual's magnitude of loading in a controlled environment and does not consider the frequency and duration of repeated loading over a prolonged time period. Given the importance of load volume it may be important to consider the combination of load magnitude, frequency and duration to better manage and prevent symptomatic PT.

Previous literature demonstrates associations between training and competition load volumes and injury.[34,35] Specifically, high training load volume (training hours/week, match/week) increases the risk of PT in adolescent male and female volleyball athletes (OR: 1.72-3.38).[36] Recent evidence demonstrates a reduction in physical activity in cohorts following traumatic knee injury surgery and chronic ankle instability compared to healthy, uninjured peers.[37,38] Physical activity (PA) and sports participation in the youth population has been

associated with numerous positive health and social behaviors,[47,50] as well as continued physical activity into adulthood.[46] Advances in wearable technology, specifically accelerometry, allow for quantification of PA metrics that provide objective insight into cumulative external loading in an individual's natural environment.[170,180] Accelerometry-based measurement of PA is considered superior to self-reported PA quantification because it removes recall error bias and is able to more objectively quantify the amount and intensity of various forms of PA.[165,176,177,215] The most common outputs from PA measurement include step counts and moderate-to-vigorous activity (MVPA), both measures that give insight into load frequency and duration.

Examining loading frequency may be especially important in better understanding overuse injuries. However, to-date, there are no studies investigating objective measures of physical activity in individuals with PT. As the pathoetiology of tendinopathy acts on a continuum,[9,10] it is possible that individuals at different stages of the continuum (i.e. asymptomatic vs. symptomatic, with or without structural pathology) may undertake different levels of activity, which may be important in the progression of the condition. Additionally, though numerous studies demonstrate that athletes with patellar tendinopathy report high-levels of activity related pain on subjective patient-reported outcomes,[203,236,245] it is unclear if these measures associate with objective measures of physical activity.

The utility of quantifying load frequency and duration metrics in this population is to obtain a more objective understanding of the associations between cumulative load volume and clinical manifestations of patellar tendinopathy (i.e. structural pathology and pain). It is possible that measuring and tracking cumulative load frequency and duration are critical pieces missing from current investigations related to the management of patellar tendinopathy. Previous literature has demonstrated that using an objective metric of cumulative load volume (discrete

biomechanical measure * load frequency measure) is effective at both predicting the onset of musculoskeletal (MSK) disease as well as distinguishing between individuals with and without MSK disease.[39,246] Maly et al. developed an approach to estimate cumulative knee loading demonstrates high reliability.[39] Based on the foundational knowledge of tendon as a mechano-responsive tissue, and growing evidence of altered real-world loading in pathological populations, we propose applying the cumulative knee loading metric to evaluate load volume measures that have previously been described to differ between individuals with and without patellar tendinopathy in traditional biomechanical assessments.[19,23,158,247]

Therefore, the primary purpose of this study was to investigate differences in load volume, magnitude, duration, and frequency measures between male athletes with and without patellar tendinopathy. We hypothesized that, compared to healthy control particitipants athletes with symptomatic PT will demonstrate patterns under-loading while athletes with asymptomatic PT will demonstrate patterns of over-loading, respectively.

METHODS

Participants

Forty-one male participants with and without patellar tendinopathy were enrolled into this study (Table 1). Participants were recruited from the local high school and university communities using approved email correspondence and public flyers. All participants were 15-28 years-old and were required to be actively participating within an organized sport setting, quantified by a Tegner Activity Scale of ≥ 5, considered to be post-pubertal, quantified by Pubertal Development Scale Stage 5 (score >12).[106,202] This study was approved by the university's institutional review board and informed consent was obtained by all participants prior to study screening.

Screening Protocol for Patellar Tendinopathy

To determine group assignment, all participants underwent a two-part screening protocol. Participants were recruited into the symptomatic PT group (SYM-PTA) if they exhibited: 1) pain ≥2/10 on the numeric rating scale (NRS) only in the patellar tendon during performance of the Single Leg Decline Squat (SLDS) test[110], as selected from a pain map diagram providing a series of pictures with various anterior knee pain locations (if bilateral pain, the "worse" limb must have been ≥ 5/10 and the contralateral limb must have been ≤ 2/10 on NRS (0-10)), and 2) ultrasonographic (US) evidence of a structural proximal patellar tendon abnormality (PTA), as defined by the presence of a hypoechoic region (≥ 2mm) and/or a maximum thickness of > 7mm, evident on both the longitudinal and transverse scans.[80,85] All US images were obtained by a single trained investigator (L.S.P.). Participants were recruited into the asymptomatic PT group (ASYM-PTA) if they were free of SLDS pain but demonstrated US evidence of a PTA. Finally, participants were recruited into the healthy control group (CON) if free of SLDS pain and PTA.

Any participants were excluded if they exhibited any of the following: 1) known neurological disorders or cardiopulmonary diseases, 2) a history of any lower extremity surgery, 3) a history of a lower extremity injury in the prior six-months, 4) an injection to the patellar tendon in the prior last three-months, 5) participation in formal rehabilitation for anterior knee pain in the prior three-months, 6) presentation of non-tendinopathic knee pain during the SLDS test (i.e. patellofemoral pain syndrome presentation), or 7) any other medical condition that would prevent them from participation in normal activities of daily living.

Patient-Reported Outcomes

The Victorian Institute of Sport Assessment-Patellar Tendon (VISA-P) questionnaire was used to quantify self-reported knee function.[203,204] The VISA-P consists of 8-items regarding the

presence of pain during various daily and sport-related activities. All participants completed this questionnaire at the time of the screening session.

Physical Activity Measurement

Data Collection

Participants were outfitted with an ActiGraph GT9X Link accelerometer (ActiGraph Corporation, Pensacola, FL), a solid-state tri-axial accelerometer with capabilities to capture, record, and store high-resolution human activity information.[209] The ActiGraph GT9X Link accelerometer was used in this study because it has been shown to be a valid and reliable accelerometer to capture objective data of steps-per-day and minutes of moderate-to-vigorous physical activity (MVPA) in young, active cohorts.[38,210,211]

Participants wore the accelerometer in a Velcro pouch via an elastic waist belt at the right anterior-superior iliac spine for a 7-day wear period. A seven-day monitoring period is an accepted duration commonly utilized in physical activity literature.[38,39,211,213] A valid wear period was considered to be at least 4 total days (3 weekdays and 1 weekend day) of at least 480 minutes (8 hours) per day. Participants were instructed to remove the accelerometer for bathing and sleeping, but to wear it at all other times throughout the day. Participants were provided with an individual docking station to connect via USB to a standard wall outlet for charging. Participants were asked to charge the accelerometer each night to ensure consistent battery life throughout daily wear periods. Feedback feature display (i.e. steps/day) on the screen of the accelerometer was disabled in order to avoid participant bias on their daily performance. To maximize the acquisition of quality data, participants were provided with an instructional and troubleshooting guide and the principal investigator's cellular phone number. Finally,

participants kept daily physical activity logs, including both exercise and sport-specific activity, during the wear period. Following the conclusion of the wear period, participants returned the accelerometer and exercise diaries, and data were inspected to ensure they met the wear requirements. If insufficient data had been obtained, participants were asked to re-wear the accelerometer for an additional 7-day wear period.

Data Analysis

The primary outcome variables for this study were average moderate-to-vigorous-activity (MVPA) per day (MVPA/day (min)), average steps-per-day (steps/d), and average steps-in-MVPA-per-day ($steps_{MVPA}$/day) during the valid wear period. The ActiGraph GT9X Link accelerometer measured accelerations in the range of +/- 8g at a 30 Hz sampling frequency in raw acquisition mode with a 60-second epoch parameter (data written to memory every 60 seconds). After participant use, data were processed and analyzed using ActiLife v6.0.0 (ActiGraph Corporation, Pensacola, FL), the proprietary actigraphy data analysis software platform of ActiGraph. Wear time validation was performed using Choi et al. (2011) algorithms,[225] which differentiates between periods of valid wear and non-wear time, and allowed the principle investigator to ensure that participants met the minimum amount of wear time per protocol. Next, Freedson Adult VM3 cut points were applied to classify physical activity into light, moderate, vigorous, and very vigorous categories based on the number of activity counts per 60-second epoch.[248] The number of steps-per-day is calculated within ActiLife software based on the vertical acceleration data measured with the GT9X Link monitor. All variables of interest were normalized to the number of valid wear days prior to analysis.

Three-Dimensional Landing Assessment

Testing Protocol

On a separate testing day following the 7-day accelerometer wear period, participants visited the laboratory for a three-dimensional biomechanical landing assessment. Participants performed a 5-minute warm-up on a stationary bicycle at a self-selected pace.

Double-Limb Jump Landing Task

Participants were provided with spandex shorts and tops. Participants wore their own athletic shoes. Participants performed five trials of a jump-landing task from a 30 cm box that was positioned 50% of the participant's height from the front edge of the force plates.[221] The participants were instructed to jump forward off the box to a double-leg landing with one foot on each force plate, and immediately perform a maximal vertical jump upon landing.[221] A minimum of one practice trial was performed; practice trials were performed until the participant and investigator ensured correct performance of the jump-landing task. A total of five jump-landing trials were collected, and the middle three trials were averaged for data analysis. If one of the middle three trials was not successful, a subsequent trial was utilized for analysis. A successful trial required the participant to leave the box with both feet at the same time, land on the force plates, and jump straight up in the air as high as possible.

Participants were outfitted with 20 retro-reflective markers bilaterally on the following bony landmarks: acromion process, anterior superior iliac spine (ASIS), greater trochanter, medial and lateral femoral condyles, medial and lateral malleoli, calcanei, and the first and fifth metatarsal heads.[219] A single marker was placed on the manubrium of the sternum and at the L4-L5 vertebral space. Rigid clusters of three or four markers was placed at the sacrum and on the thigh, shank, and foot segments bilaterally. A static trial was captured with the participant

standing with arms positioned at 90° of shoulder abduction to estimate the location of the landmarks needed to calculate joint centers.[219] After the static trial, the single markers on the foot, malleoli, femoral condyles, and greater trochanters were removed.

Data Acquisition

Three-dimensional kinematic data were collected using a ten-camera motion capture system (Vicon Motion Systems, Centennial, CO, USA) sampled at 120Hz and filtered using a fourth-order low-pass Butterworth filter with a 12Hz cutoff frequency. Kinetic data were sampled at 1200Hz using two floor embedded force plates (Bertec Corporation, Columbus, OH, USA). Knee and ankle joint center coordinates were defined as the centroid between the medial and lateral condyles and malleoli identified during the static trail. Hip joint center coordinates were estimated from the coordinates of the L4-5, right ASIS, and left ASIS markers using the Bell method.[222] Reference frames for the foot, tibia, and femur were defined based on 3D-coordinates and segments as follows: 1st and 5th metatarsal heads, ankle joint center, and calcaneus (foot); medial and lateral malleoli, knee and ankle joint centers, and shank (tibia); medial and lateral femoral condyles, knee and hip joint centers, and thigh (femur). Joint angles were defined based on the position of the distal segment relative to the proximal segment using a Cardan angle sequence in the following order of rotation: sagittal (y-axis'), frontal (x-axis'), and transverse (z-axis').

Data Processing & Reduction

Lower extremity biomechanics for the involved (SYM & ASYM) and dominant (CON) limbs were evaluated during the descending phase of the jump-landing task, which was defined as the interval from initial contact (IC) to peak knee flexion during the stance phase.[223] IC was defined as the time point when the vertical ground reaction force (vGRF) exceeded 10N.[223] The

stance phase was defined as the time from IC until toe off (vGRF <10 N). Peak knee flexion was defined as the maximal flexion angle during the stance phase and was used to determine the end of the descending loading phase (IC to time of peak knee flexion angle).

Ground reaction force data and processed segment data were used to calculate net internal sagittal and frontal plane knee and hip joint moments using inverse dynamics procedures.[146] Patellar tendon force (F_{PT}) was estimated using previously defined methodology of Nisell and Ekholm, through with F_{PT} is calculated by dividing the internal knee extension moment by the patellar tendon moment arm.[160] PT moment arms were calculated as a function of knee joint angles using the methodology of Herzog and Read.[144]

F_{PT} impulse ($F_{PT}I$) and internal knee extension moment impulse (KEMI) were calculated as the area under the F_{PT} and KEM curves, respectively, during the descending phase of the landing task. Knee power (J/s) was calculated as the product of the internal sagittal plane knee moment (Nm/kgm)*knee flexion velocity (°/millisecond). Negative knee work (KW) (J) was calculated as the negative area under the knee power curve during the descending phase of the landing task.

Loading volume variables were estimated based on the methodology of Maly et al.[39] However, because our biomechanical outcome variables were assessed during a dynamic landing task, the number of steps-per-day in MVPA ($steps_{MVPA}$) was utilized for this study instead of the total steps/day (which includes steps in the "light" physical activity category). MVPA has been described previously as including activity ranging from weight training to jogging to competitive sport.[249] Average $steps_{MVPA}$/day was divided by two, since the accelerometer captures steps for both limbs. Loading volume variables included: patellar tendon load (cF_{PT}), cumulative patellar tendon force impulse ($cF_{PT}I$), internal knee extension moment impulse (cKEMI), knee power

(cKP), and knee work (cKW). The following equation was utilized, using the variable 'cF_{PT}' as an example:

$$\textbf{cF}_{\textbf{PT}}\textbf{ load estimation: } \int_a^b F_{PT}(t) * dt \times \left(\frac{steps_{MVPA}}{day}\ for\ test\ limb\right)$$

where cF_{PT} is the cumulative patellar tendon force per day estimation; $F_{PT}(t)$ is the mean patellar tendon force (F_{PT}) across the three jump-landing trials at time (t) from a) initial ground contact, to b) peak knee flexion angle.

Statistical Analysis

Descriptive characteristics (means, standard deviations, 95% confidence intervals (CI)) were calculated for all dependent variables (Table 1). Physical activity and biomechanical variables were compared between the three groups (CON, ASYM-PTA, SYM-PTA) using a one-way analysis of variance (ANOVA). Tukey post-hoc tests for pairwise comparisons of means for each dependent variable were performed for significant findings from each ANOVA model. Mean differences between groups and associated 95% confidence intervals were calculated. Cohen's *d* effect sizes were used to evaluate the magnitude of between group differences for loading volume variables, classified as weak ($d \leq 0.2$), small ($d = 0.2$-0.5), moderate ($d = 0.5$-0.8), or large ($d \geq 0.8$).[226] Pearson product-moment correlations were utilized to examine the relationship between objective physical activity measures and VISA-P score. Statistical significance was set *a priori* at $\alpha < 0.05$. All statistical analyses were completed in SPSS v22 (IBM Inc., Armonk, New York, USA).

Table 1: Descriptive characteristics of the study population (mean ± sd)

	Healthy Control (n=14)	Asymptomatic Tendinopathy (n=14)	Symptomatic Tendinopathy (n=13)
Age (yrs)	19.64 ± 1.60	21.00 ± 1.96	19.62 ± 1.61
Height (m)	1.84 ± 0.09	1.84 ± 0.07	1.82 ± 0.05
Mass (kg)	79.91 ± 12.95	81.63 ± 13.03	83.46 ± 5.12
Tegner Activity Scale (0-10)	8.00 ± 0.88	8.00 ± 1.04	8.00 ± 1.00
Pubertal Development Scale (0-12)	11.57 ± 0.65	11.86 ± 0.53	11.39 ±0.87
VISA-P (0-100)	97.64 ± 3.41	94.07 ± 7.85	76.15 ± 13.37*^

*: statistically significant difference than CON group (p<0.001, MD: -21.49 (-29.97, -13.01))
^: statistically significant difference than ASYM group (p<0.001, MD: -17.91 (-26.40, -9.44))

RESULTS

Demographic data are presented in Table 1. No significant differences in height, mass, or age were observed between groups (p > 0.05). The VISA-P score was significantly lower in the SYM-PTA group compared to both the ASYM-PTA and CON groups (p<0.001), and the mean difference exceeded the MCID (13 points) for this subjective outcome measure.[204]

Results from the load volume analyses are presented in Table 3. Participants with SYM-PTA demonstrated lesser load volume than CON for all variables (cF_{PT}, $cF_{PT}I$, cKEMI, cKP, cKW) (p<0.05). There were no significant differences in load volume between the SYM-PTA and ASYM-PTA groups or the ASYM-PTA and CON groups (p>0.05). Mean differences and Cohen's d effect sizes are presented in Table 4. The magnitude of the effect for SYM-PTA compared to CON was considered to be strong and significant for cF_{PT} (d =0.98), $F_{PT}I$ (d = 1.09), KEMI (d = 1.09), cKP (d = 10.4), and KW (d = 1.14) (Table 4).

There were no significant differences between the three groups for the load frequency (steps/day, $steps_{MVPA}$/day) or duration (MVPA/day) metrics (p>0.05) (Table 2). However, there was a non-significant trend of fewer steps/day, $steps_{MVPA}$/day, and MVPA/day in the SYM-PTA

group compared to both the CON and ASYM-PTA groups (Table 2). Total wear time was not significantly different between groups (p=0.205). All group comparisons were conducted with and without controlling for total wear time, and neither model demonstrated statistical significance (p>0.05).

Results from the biomechanical load magnitude analysis are presented in Table 5. The SYM-PTA group demonstrated significantly less load magnitude than the CON (p<0.01) and ASYM-PTA (p>0.05) groups for all variables ($F_{PT}I$, KEMI, KW). There were no statistically significant differences in these baseline biomechanical load magnitude variables between the CON and ASYM-PTA groups (p>0.05). Mean differences and Cohen's d effect sizes are presented in Table 6. The magnitude of the effect for SYM-PTA compared to CON was considered to be strong and significant for $F_{PT}I$ (d = 1.40), KEMI (d = 1.42), and KW (d = 1.53).

Table 2: Descriptive characteristics for study population for load frequency and duration metrics (mean ± sd, 95% CI)

	Healthy Control (n=14)		**Asymptomatic Tendinopathy (n=14)**		**Symptomatic Tendinopathy (n=13)**	
	Mean ± SD	*95% CI*	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Total steps/day	11,195 ± 1,803	10,154, 12,237	10,143 ± 2,646	8,615, 11,671	10,250 ± 2942	8,472, 12,028
Steps in MVPA/day	7,028 ± 3,329	5,106, 8,951	5,977 ± 2,723	4,405, 7,549	5,487 ± 1716	4,450, 6,524
Time in MVPA/day (min)	102.42 ± 20.73	90.45, 114.40	97.60 ± 37.67	75.85, 119.36	94.54 ± 29.10	76.96, 112.13
Time in MVPA/day (%)	13.27 ± 3.41	11.30, 15.24	11.80 ± 5.57	8.58, 15.02	12.76 ± 4.43	10.08, 15.44
Total wear time (min)	5,298 ± 968	4,739, 5,859	5,217 ± 881	47,08, 5,725	4,598 ± 1,375	4,700, 5,397
Valid wear days	6.58 ± 0.51	6.28, 6.87	6.43 ± 0.85	5.94, 6.92	5.92 ± 0.31	5.25, 6.60

**Note: the values reported are unadjusted for total wear time. No statistical difference between groups for total wear time (p=0.205)*
**No statistically significant differences between groups, both with and without controlling for total wear time (minutes) (p>0.05).*

Table 3: Descriptive characteristics (mean ± sd, 95% CI) for load volume variables (based on # of $steps_{MVPA}$ and involved limb biomechanics during the double-limb landing task).

Table 3a:

	Cumulative PTF (BW)		**Cumulative PTF Impulse (BW*ms)**		**Cumulative KEM Impulse (Nm*ms)**	
Group	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Healthy Control (n=14)	$-12.3e^3 \pm 6.17e^3$	$-15.9e^3$, $-8.74e^3$	$-2.27e^6 \pm 1.24\ e^6$	$-2.99e^6$, $-1.56\ e^6$	$-10.7e^4 \pm 5.96e^4$	$-14.1e^4$, $-7.24e^4$
Asymptomatic Tendinopathy (n=14)	$-9.97e^3 \pm 4.63e^3$	$-12.5e^3$, $-7.45e^3$	$-1.76e^6 \pm 0.880e^6$	$-2.26e^6$, $-1.25e^6$	$-8.15e^4 \pm 4.04e^4$	$-104.9e^4$, $-5.82e^4$
Symptomatic Tendinopathy (n=13)	**$-7.81e^3 \pm 1.94e^3$***	$-8.98e^3$, $-6.64e^3$	**$-1.22e^6 \pm 0.566e^6$***	$-1.56e^6$, $-8.75e^5$	**$-5.70e^4 \pm 2.56e^4$***	$-7.25e^4$, $-4.15e^4$

Table 3b:

	Cumulative Knee Power (J*ms)		**Cumulative Knee Work (J)**	
Group	*Mean ± SD*	*95% CI*	*Mean ± SD*	*95% CI*
Healthy Control (n=14)	$-6.71e^3 \pm 3.46e^3$	$-8.71e^3$, $-4.72e^3$	$-5.56e^5 \pm 3.38e^5$	$-7.51e^5$, $-3.60e^5$
Asymptomatic Tendinopathy (n=14)	$-5.31e^3 \pm 2.59e^3$	$-6.80e^3$, $-3.81e^3$	$-3.70e^5 \pm 1.72e^5$	$-4.70e^5$, $-2.71e^5$
Symptomatic Tendinopathy (n=13)	**$-4.03e^3 \pm 1.18e^3$***	$-4.74e^3$, $-3.32e^3$	**$-2.64e^5 \pm 1.25e^5$^**	$-3.39e^5$, $-1.88e^5$

**statistically significant difference than the healthy control group (p<0.05)*
^ statistically significant difference than the healthy control group (p<0.01)

Table 4: Group comparisons for load volume variables (based on # of $steps_{MVPA}$ and involved limb biomechanics during the double-limb landing task).

Table 4a:

	Cumulative Patellar Tendon Force (BW)			Cumulative Patellar Tendon Force Impulse (BW*ms)			Cumulative Knee Extension Moment Impulse (Nm*ms)		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	$-4.49e^3$	$-8.76e^3$, $-.21e^3$	0.98	$-10.57e^5$	$-19.44e^5$, $-1.69e^5$	1.09	$-4.99e^4$	$-9.17e^4$, $-.80e^4$	1.09
CON vs. ASYM	$-2.33e^3$	$-6.52e^3$, $1.87e^3$	0.44	$-5.17e^5$	$-13.88e^5$, $3.54e^5$	0.48	$-2.54e^4$	$-6.64e^4$, $1.57e^4$	0.50
ASYM vs. SYM	$-2.16e^3$	$-6.43e^3$, $2.11e^3$	0.64	$-5.40e^5$	$-14.27e^5$, $3.48e^5$	0.73	$-2.45e^4$	$-6.63e^4$, $1.73e^4$	0.72

Table 4b:

	Cumulative Knee Power MVPA (J*ms)			Cumulative Knee Work MVPA (J)		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	$-2.68e^3$	$-5.13e^3$, $-.23e^3$	1.04	$-2.92e^5$	$-5.11e^5$. $-.74e^5$	1.14
CON vs. ASYM	$-1.41e^3$	$-3.81e^3$, $1.00e^3$	0.46	$-1.86e^5$	$-4.00e^5$, $.29e^5$	0.69
ASYM vs. SYM	$-1.27e^3$	$-3.73e^3$, $1.18e^3$	0.63	$-1.07e^5$	$-3.26e^5$, $1.12e^5$	0.64

Group Legend: CON=healthy control, ASYM=asymptomatic tendinopathy, SYM=symptomatic tendinopathy
Note: The group listed first is the referent group for each mean comparison.

Table 5: Group comparisons for load magnitude variables for the involved (SYM & ASYM) and dominant (CON) limbs during the double limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)		**Patellar Tendon Force Impulse (BW*ms)**		**Negative Knee Work (J/kg)**	
	Mean	*95% CI*	*Mean*	*95% CI*	*Mean*	*95% CI*
Healthy Control (n=15)	-29.98 ± 4.91	-32.70, -27.25	-639.20 ± 102.82	-696.14, -582.26	-156.37 ± 34.45	-175.45, -137.29
Asymptomatic Tendinopathy (n=15)	-27.13 ± 5.91	-30.41, -23.85	-583.34 ± 129.80	-655.22, -511.46	-126.58 ± 26.81	-141.43, -111.74
Symptomatic Tendinopathy (n=13)	**-21.19 ± 7.28**[*#]	-16.79, -25.59	**-450.81 ± 160.57**[*#]	-547.84, -353.78	**-98.72 ± 40.67**[*#]	-123.30, -74.15

*statistically significant difference compared to CON group (p<0.01)
#statistically significant difference compared to ASYM group (p<0.05)

Table 6: Effect size calculations for group comparisons for load magnitude variables for the involved (SYM & ASYM) and dominant (CON) limbs during the double limb landing task.

	Internal Knee Extension Moment Impulse (Nm*ms)			**Patellar Tendon Force Impulse (BW*ms)**			**Negative Knee Work (J/kg)**		
Group Comparisons	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*	Mean Difference	95% CI	Cohen's *d*
CON vs. SYM	-8.79	-14.37, -3.31	1.42	-188.39	-309.80, -66.97	1.40	-57.65	-89.10, -26.19	1.53
CON vs. ASYM	-2.85	-8.22, 2.53	0.52	-55.86	-172.86, 61.14	0.48	-29.79	-8.22, 2.53	0.97
ASYM vs. SYM	-5.94	-11.52, -0.36	0.90	-132.53	-253.94, -11.12	0.91	-27.86	-11.52, -0.36	0.81

Legend: SYM: symptomatic tendinopathy, ASYM: asymptomatic tendinopathy, CON: healthy control
Note: The group listed first is the referent group for each mean comparison.

DISCUSSION

Our most important finding was that load volume differences were observed across groups. Specifically, the symptomatic participants displayed significantly lesser load volume compared to the healthy control participants, clearly demonstrating an under-loading profile. In contrast to our hypothesis, load volume was not greater in the asymptomatic participants compared to either the healthy control or symptomatic participants. Thus, there was no pattern of over-loading observed for those with asymptomatic PT. Patterns of reduced load volume in our symptomatic participants appear to be driven by lesser biomechanical load magnitude, as $F_{PT}I$, KEMI, and KW were all reduced in symptomatic compared to healthy control participants; however, there were no differences in measures of load frequency (steps/day) or duration (MVPA/day). To the best of our knowledge, this is the first study to objectively measure load volume, frequency, and duration metrics in individuals with patellar tendinopathy, and the results reinforce the multifaceted nature of this injury condition.

The first key finding of this study is that individuals with SYM-PTA demonstrate both lesser magnitude and volume of involved-limb patellar tendon loading and knee energy absorption than both ASYM-PTA and CON groups. The magnitude of loading we observed is consistent with those of Sorenson et al (2010), which found 29% less mechanical energy absorption in individuals with tendinopathy compared to healthy controls during landing.[19] Additionally, Bisseling et al. (2007) demonstrated a load-avoiding landing strategy in athletes with symptomatic tendinopathy, including reduced knee joint power, work, and lower peak knee moments.[22] In the present study, we saw large effects ($d > 0.8$) between the CON and SYM groups. Likely driven by pain, this movement pattern reflects a load-avoidance behavior, whereby individuals reduce extensor mechanism loading and absorption of mechanical energy

during the descending phase of the landing task. Mechanical power and work reflect the interaction of load and displacement, which are highly relevant to the sagittal plane demand on the patellar tendon during jumping maneuvers. Chronic reduction in energy absorption and eccentric stimulus to the tissue may reduce the effectiveness of stretch-shortening cycle and the ability of the patellar tendon to appropriately perform force transmission, which may lead to other compensatory movement strategies.

Of particular interest from our analysis is the quantification of the magnitude and rate of load across the patellar tendon (PTF and $F_{PT}I$ variables), both discretely and cumulatively, demonstrating lesser tissue loading in the symptomatic group. Edwards et al. (2012) found that normalized peak patellar tendon forces were significantly greater than peak vertical ground reaction forces (VGRF) during the horizontal phase of a stop-jump task, suggesting that using VGRF to represent patellar tendon load may underestimate the specific load across the tissue.[158] Our results illustrate that biomechanical under-loading observed in a laboratory setting can be perpetuated in an individual's natural environment. For individuals following soft tissue or joint injuries, chronic under-loading may fail to provide the mechanical stimulus needed to maintain tissue homeostasis within its envelope of function.[64,250]

A novel aspect of the current study is the use of cumulative patellar tendon and knee joint loading estimations, which blend biomechanical (load magnitude) and physical activity (load frequency and duration) metrics to more objectively quantify load volume. Driven by group differences in the magnitude of biomechanical loading, SYM-PTA participants demonstrated significantly less loading volume than both ASYM-PTA and CON groups, suggesting chronic under-loading in individuals with symptomatic patellar tendinopathy. Regardless of the lack of differences in loading frequency and duration (average steps/day and MVPA minutes/day)

between groups, lower discrete loading magnitudes on the involved limb in the symptomatic group still resulted in cumulative under-loading when extrapolated into the individual's real-world environment. Over time, chronic under-loading will lead to reduced tissue capacity through stress-shielding, and limit the adaptive potential to the tissue when exposed to high loading demands of sport.[9,251] The use of the cumulative load estimation emphasizes the importance of acknowledging comprehensive load volume, particularly in pathologic populations.[39] Future work should seek to develop feasible, implementable strategies to identify changes in physical activity that may be reflective of trends in either over- or under-loading that may result in deleterious outcomes.

Interestingly, there were no differences in loading frequency and duration between our three groups. These findings were in contrast to our hypotheses that the SYM group would demonstrate lesser and the ASYM group would demonstrate greater loading frequency and duration than the CON group, respectively. It is well-accepted in the literature that the development and progression of patellar tendinopathy in athletes is often related to the mismanagement of external load over time.[9–11,90,251] Though multifactorial, the load-related pathoetiology of tendinopathy stems from a discrepancy between the tendon tissue's load capacity and imposed external load.[10,90] Our findings may have important clinical implications in how clinicians design, prescribe, and manage load exposure in athletes at various stages of tendinopathy continuum.

A study conducted by Visnes and Bahr (2013) tracked training volume prospectively for four-years via self-reported training diaries in young elite volleyball players, and found that the development of symptomatic patellar tendinopathy associated with higher overall training characteristics, specifically number of hours and matches played.[36] It is possible that the

specificity of the activity-related measures to the sport of volleyball (hours and matches played) used by Vises and Bahr better captured the type of load related to tendinopathy development than general measures of steps- or MVPA- per-day used in the current study. Tendinopathy is most prevalent in athletes participating in jumping sports,[5] and the risk of patellar tendinopathy is associated with jumping performance.[5,36,140,156] However, while there is evidence of sex and inter-individual differences in jumping frequency,[252] little is known on how jumping frequency may influence the risk or progression of patellar tendinopathy. In light of current gaps in existing evidence and the current study's findings, an important next step in load management should include serial monitoring of sport-specific movements, such as tracking jump-counts over time, which may be more helpful in understanding the effect of real-world load volume on patellar tendinopathy.

Due to the cross-sectional nature of the current study, we are unable to determine if the onset of the structural pathology and/or symptoms in the ASYM-PTA and SYM-PTA groups was associated with a prior change in physical activity, such as training volume or intensity. At the time of enrollment in this study, all participants were participating in sport activity. We hypothesized that individuals with symptoms would self-restrict their physical activity due to pain-avoidance behavior. However, despite worse VISA-P scores, the SYM-PTA group did not differ from the other groups in physical activity. Additionally, VISA-P score was not correlated with either physical activity measure (p>0.05). VISA-P scores in the SYM-PTA group (76.15 ± 13.37 points) were comparable to symptomatic cohorts of similar ages and activity levels in previous studies.[19,22,140,253] This finding may have important clinical implications, as it suggests that individuals who report high levels of pain-related disability do not necessarily limit their physical activity. Additionally, our results indicate that patient-reported outcomes may not

accurately reflect the influence of pain on physical activity if used in isolation. Longitudinal studies that regularly monitor both physical activity and patient-reported function are needed to better understand the effects of tendinopathy on sport participation. Proper education for patients and athletes that acute spikes in load, particularly sport-specific loads that exceed tissue capacity, may increase the risk of negative progression along the tendinopathic continuum is important.[165,171,173]

On average, each group in the current study met the well-accepted recommendation of 10,000 steps/day for adults.[254] Studies that utilize college-aged participants acknowledge that these individuals are highly active due to the nature of their daily activities, such as walking to class. Using similar 7-day monitoring procedures, several previous studies have observed healthy college-aged participants to average between approximately 8,800 and 10,000 steps/day,[37,38,255] with Mestek et al. (2008) specifically observing an average 10,027 ± 3,535 steps/ day in a cohort of 44 college males.[255] While comparable, all participant groups in our study completed more steps/day than these previous studies, which may be due to our inclusion criteria of a minimum Tegner Activity Scale score (5/10), indicating athletic participation at least at the recreational level. Additionally, all participant groups were well over the 150 minutes of MVPA activity per week recommended by the American College of Sports Medicine.[256]

These combined findings suggest that even individuals with symptomatic patellar tendinopathy do not significantly limit the frequency or duration of physical activity compared to their healthy counterparts. Additionally, there was no evidence of over-loading in asymptomatic participants as neither load magnitude, volume, duration, or frequency was higher in this group. In fact, there is a non-significant trend for under-loading relative to healthy control participants. These findings suggest that reducing load may not be the ideal treatment for asymptomatic

individuals with structural patellar tendon abnormalities. Rather, progressive loading protocols designed to increase the capacity of healthy tendon tissue may be critical for this cohort of tendinopathy patients to prevent the development of symptoms. Future research should seek to study the effects of using comprehensive strategies, including both load magnitude and volume measurements, to determine how to appropriate manage load in individuals at different stages of the tendinopathic continuum.

LIMITATIONS

This study is not able to offer cause-and-effect evidence that biomechanical movement strategies or physical activity levels preceded the development of tendinopathy or result in response to the pathologic condition. There are currently no prospective longitudinal studies that assess biomechanical profiles and adaptations around the initial development of patellar tendinopathy. We are also unable to account for other factors that may have influenced the participants' physical activity levels during the seven-day monitoring period, such as motivation, academic demand, or environmental factors related to their sport, or the specific types of activity that may have differed between groups. There are obvious limitations to extrapolating seven-days of physical activity measurement as a reflection of an individuals' actual physical activity over a protracted time-frame. However, a seven-day monitoring period is an accepted duration commonly utilized in physical activity literature, and has been shown to improve reliability and decreased variability of objective physical activity data.[38,39,211,213] Finally, we did not take a daily pain assessment during the seven-day physical activity monitoring week, so we are unable to determine if daily fluctuations in pain may have influenced physical activity participation in the symptomatic group.

CONCLUSIONS

Male athletes with symptomatic patellar tendinopathy do not differ in general measures of physical activity frequency and duration compared to both asymptomatic patellar tendinopathy and healthy control participants, despite higher self-reported pain levels during functional and sport-related tasks. However, lesser magnitudes of patellar tendon force and energy absorption across the knee joint during landing in symptomatic individuals results in cumulative under-loading over a seven-day monitoring period. This study's findings highlight the importance of a comprehensive approach to load monitoring in individuals with patellar tendinopathy, including biomechanical movement profiles, loading volume, and patient-reported outcomes.

CHAPTER 7: MANUSCRIPT 3

Landing Biomechanics are Not Immediately Altered by an Isometric Patellar Tendon Loading Exercise Protocol in Male Athletes with Patellar Tendinopathy

INTRODUCTION

Tendon adaptation occurs through mechanotransduction, the physiological process by which the body translates mechanical load into a cellular response that leads to structural change.[12,257] Mechanotherapy describes how load can be manipulated to influence the mechano-transductive events within the tissue.[12] As such, load-based exercise is considered the gold-standard of tendon rehabilitation.[10,17,258] Eccentric-based exercise protocols have traditionally been utilized in the treatment of chronic tendinopathies.[13–15,66] In the degenerative stage of chronic tendinopathy, eccentric exercise has been shown to be effective at reducing pain,[13–15,186] increasing force, stiffness,[259] and Young's elastic modulus,[187] decreasing tendon thickness,[79] and improving self-reported function.[79]

Not all tendinopathy patients respond positively to eccentric exercise.[77,260–262] Specifically, athletes continuing to participate in activity while managing patellar tendinopathy, the capacity to tolerate high sporting loads plus heavy eccentric exercise treatments may be limited.[90,260] Current evidence suggests that loading interventions, including type and dosage of exercise, should be prescribed based on where a tendon falls on the continuum of pathology.[9] Cook et al. (2016) suggests that 'phenotyping' patients based on the presence of pain, structural

abnormalities, and self-reported dysfunction may help clinicians direct patient-centered treatments to improve short- and long-term patient outcomes.[10]

Emerging evidence supports the use of isometric exercise for individuals with symptomatic patellar tendinopathy. Sub-maximal isometric exercise improved pain and self-reported function and modulates inhibition in adults with symptomatic patellar tendinopathy,[16–18] and demonstrated excellent patient compliance and tolerance when implemented in-season.[16] Isometric exercise is thought to be a more appropriate treatment option than eccentrics for painful, competing athletes because it promotes heavy loading to stimulate the mechano-transductive processes needed for tendon adaptation without being provocative,[16,18,90] as often occurs with heavy eccentric training.[13,261]

We believe that interventions able to modify symptoms may allow for changes in biomechanical movement profiles. Specifically, in those with symptomatic patellar tendinopathy, an intervention that is able to reduce pain may allow the individual to normalize their loading patterns, as athletes with symptomatic patellar tendinopathy are shown to display under-loading profiles.[19,21] Thus, a load-based exercise intervention may provide three key benefits. First, isometric loading increases the volume of stimulus placed on the patellar tendon in a given session, which may have positive mechano-transductive effects on the surrounding aligned fibrillar tissue.[10,80,94] Second, isometric exercise acutely reduces pain and decreases cortical inhibition.[17] And finally, a reduction in pain may allow for the individual's biomechanical profile to normalize, minimizing the amount of under-loading exhibited during high-energy tasks. Consequently, the isolated isometric exercise protocol may encourage greater tendon loading during dynamic tasks, which may be an even more powerful and systematic way to improve the volume of tissue loading over time during sport-related activities. By selecting the appropriate

type of load-based intervention, particularly in early stages of rehabilitation, progressively building tissue capacity may be more tolerable to patients and therefore improve the effectiveness of other interventions, such as neuromuscular re-education and movement-retraining.[82,94,188,189]

The effects of an exercise protocol that targets tendon-specific loading on movement profiles has not yet been investigated. Therefore, the purpose of this study was to determine the acute effects of an acute bout of patellar tendon isometric loading exercise on involved limb landing biomechanics in individuals with symptomatic and asymptomatic patellar tendinopathy. A secondary purpose was to determine if individuals with symptomatic patellar tendinopathy demonstrated changes in pain following the isometric loading exercise. Determining whether isometric loading acutely changes movement biomechanics may provide an important next step in rehabilitation paradigms for tendinopathy as a method to promote load-tolerance and stimulate positive mechano-transductive responses in individuals with tendon pathology

METHODS

This study utilized a single-blinded, randomized cross-over trial with two intervention conditions. Twenty-eight male participants with patellar tendinopathy were enrolled into this study (Table 1). Participants were recruited from the local high school and university communities using approved email correspondence and public flyers. All participants were 15-28 years-old and were required to be actively participating within an organized sport setting, quantified by a Tegner Activity Scale of ≥ 5, a self-reported questionnaire in which the participant identifies the highest level of competition and/or physical activity currently performing.[235] Additionally, all participants were considered to be post-pubertal, quantified by

Pubertal Development Scale Stage 5 (score >12).[106,202] This study was approved by the university's institutional review board and informed consent was obtained by all participants prior to study screening.

Screening Protocol

Group Assignment

To determine group assignment, all participants underwent a two-part screening protocol. Participants were recruited into the symptomatic PT group (SYM-PTA) if they exhibited: 1) pain ≥2/10 on the numeric rating scale (NRS) only in the patellar tendon during performance of the Single Leg Decline Squat (SLDS) test[110], as selected from a pain map diagram providing a series of pictures with various anterior knee pain locations (if bilateral pain, the "worse" limb must have been ≥ 5/10 and the contralateral limb must have been ≤ 2/10 on NRS (0-10)), and 2) ultrasonographic (US) evidence of a structural proximal patellar tendon abnormality (PTA), as defined by the presence of a hypoechoic region (≥ 2mm) and/or a maximum thickness of > 7mm, evident on both the longitudinal and transverse scans.[80,85] All US images were obtained and processed by a single trained investigator (L.S.P.). Participants were recruited into the asymptomatic PT group (ASYM-PTA) if they were free of SLDS pain but demonstrated US evidence of a PTA.

Any participants were excluded if they exhibited any of the following: 1) known neurological disorders or cardiopulmonary diseases, 2) a history of any lower extremity surgery, 3) a history of a lower extremity injury in the prior six-months, 4) an injection to the patellar tendon in the prior last three-months, 5) participation in formal rehabilitation for anterior knee pain in the prior three-months, 6) presentation of non-tendinopathic knee pain during the SLDS

test (i.e. patellofemoral pain syndrome presentation), or 7) any other medical condition that would prevent them from participation in normal activities of daily living.

Patient-Reported Outcomes

The Victorian Institute of Sport Assessment-Patellar Tendon (VISA-P) questionnaire was used to quantify self-reported knee function.[203,204,236] The VISA-P consists of 8-items regarding the presence of pain during various daily and sport-related activities and has demonstrated excellent test-retest and interrater reliability.[236] All participants completed this questionnaire at the time of the screening session.

Quadriceps Maximum Voluntary Isometric Contraction Assessment

Quadriceps strength was assessed by measuring the individual's maximum voluntary isometric contraction (MVIC) during the screening session to avoid any confounding effect of performing the baseline MVIC just prior to the intervention period.

MVIC was assessed on the HUMAC Norm Dynamometer (CSMi, Stoughton, MA) on the involved limb. All participants were positioned on the dynamometer with the test limb flexed to 60°, consistent with previous studies investigating the effects of isometric loading in a patellar tendinopathy cohort,[17,18] and to minimize added compressive forces across the anterior knee at greater knee flexion angles (i.e. 90°) that may instigate pain during contraction.[141,145] The thighs, hips and torso were firmly stabilized with straps, and the arms folded across the torso to isolate the contribution of the quadriceps muscle without extremity movement. The lever arm was adjusted so that the ankle strap was 2 finger widths (~3 centimeters) proximal to the lateral malleolus. The knee was positioned so that the lateral femoral epicondyle was aligned with the rotational axis of the dynamometer. Measurements of chair position (distance of seat-back

position, ankle strap position on lever arm) were recorded to ensure consistency between all subsequent assessments using the HUMAC system.

A series of graded submaximal warm-up isometric contractions were performed at 25%, 50%, and 75%, respectively, of the participant's perceived maximal effort but not recorded. For the MVIC testing, participants were instructed to "kick as hard and as fast as they can" into the dynamometer and to maintain maximal effort for approximately two seconds. The investigator provided standardized verbal encouragement for each trial, and participants received real-time visual feedback of their torque production on a computer monitor display directly in front of the dynamometer. Three MVIC trials were collected. If for a given trial, the participant was able to produce torque greater than 10% of the previous trial, this trial was repeated. The peak torque (N*m) of the MVIC from the three trials was recorded, and from the average of these trials, 70% +/- 5% MVIC was calculated for the isometric intervention.

Pre-Intervention Testing Protocol

Double-Limb Jump Landing Task

Participants performed a 5-minute warm-up on a stationary bicycle at a self-selected pace, after which they completed the Single Leg Decline Squat (SLDS) and rated their knee pain (NRS: 0-10). Participants were then outfitted with 20 retro-reflective markers bilaterally on the following bony landmarks: acromion process, anterior superior iliac spine (ASIS), greater trochanter, medial and lateral femoral condyles, medial and lateral malleoli, calcanei, and the first and fifth metatarsal heads.[219] A single marker was placed on the manubrium of the sternum and at the L4-L5 vertebral space. Rigid clusters of three or four markers was placed at the sacrum and on the thigh, shank, and foot segments bilaterally. A static trial was captured with the

participant standing with arms positioned at 90° of shoulder abduction to estimate the location of the landmarks needed to calculate joint centers.[219] After the static trial, the single markers on the foot, malleoli, femoral condyles, and greater trochanters were removed.

Participants were provided with spandex shorts and tops and wore their own athletic shoes. Participants performed five trials of a jump-landing task from a 30 cm box that was positioned 50% of the participant's height from the front edge of the force plates.[221] The participants were instructed to jump forward off the box to a double-leg landing with one foot on each force plate, and immediately perform a maximal vertical jump upon landing.[221] A minimum of one practice trial was performed; practice trials were performed until the participant and investigator ensured correct performance of the jump-landing task. A total of five jump-landing trials were collected, and the middle three trials were averaged for data analysis. If one of the middle three trials was not successful, a subsequent trial was utilized for analysis. A successful trial required the participant to leave the box with both feet at the same time, land on the force plates, and jump straight up in the air as high as possible.

Randomization Procedure

Randomization for the cross-over was completed by a trained research assistant using random number generating software (Random Number Generator, www.Stattrek.com, 2017) prior to the start of the study. The research assistant generated a randomization sequence for 32 participants (16 for each group) to account for potential drop-outs and produced a set of sequentially numbered opaque sealed envelopes (SNOSE) with no external markings,[208] each with a piece of paper enclosed indicating which intervention will occur at each of the two testing session. These envelopes remained sealed until the participant's eligibility was verified. The

number on the outside of the envelope will be the participant's randomization study ID. The principle investigator (PI) remained blinded to the knowledge of which intervention condition was delivered at each testing session throughout the entirety of data collection and reduction.

Intervention Protocol

For the intervention protocol, the participant was positioned in the HUMAC chair at the same position used during the screening session. Once the participant was appropriately positioned, the PI (L.S.P.) stepped out of the laboratory to remain blinded to the intervention (isometric or sham-control) for each session. Blinding of the PI was conducted in order to remove any potential bias that the PI's knowledge of the condition may have on collection and analysis of post-intervention pain and biomechanics assessments. A trained research assistant delivered the intervention. The interventions were only applied to the involved limb.

Isometric Condition

The isometric protocol was selected based on the work of Rio et al. (2015), which demonstrated acute reductions in pain and improvements in quadriceps strength in individuals with patellar tendinopathy.[17] Instructions were given to the participant prior to the beginning of the isometric condition using a standard script. The participant performed 5-sets of a 45-second isometric quadriceps contraction at 60° of knee flexion at 70% of their maximum voluntary isometric contraction (MVIC). During the 45-second quadriceps contraction, the participant was provided with visual biofeedback via the HUMAC Norm software on a computer screen positioned directly in front of the chair; this involved a real-time display of their torque output (green) and +/- 5% error lines (purple with target zone in black). The participant was instructed

to produce a level of quadriceps torque that kept the torque output line as close the target line as possible and always between the two error lines. Following each 45-second isometric contraction, the participant had 2-minutes of rest, during which their limb remained positioned passively at 60° knee flexion. This procedure was completed for 5 sets.

Sham-Control Condition

A sham-control intervention was utilized to avoid any participant-bias that the isometric condition was the experimental condition, as this perception could bias their performance on the post-intervention SLDS pain and biomechanics assessments. Participants were positioned in the identical position in the HUMAC. A sham transcutaneous electric nerve stimulator (TENS) unit (Empi Select™ TENS Device; Empi, Inc., St. Paul, MN, USA) was utilized as a sham intervention. Two (2" x 2") electrodes were placed on the medial and lateral side of the involved limb patellar tendon, respectively, and connected via a lead wire to the sham-TENS unit which was held and controlled by the trained research assistant. Instructions were given to the participant prior to the beginning of the sham-control condition using a standard script. The same time parameters used in the isometric condition were used for the sham-control condition.

Post-Intervention Testing Protocol

Immediately following the conclusion of the intervention protocol, participants repeated the same pre-intervention protocol assessments using identical methodology as described above in the following order: 1) SLDS pain assessment and 2) Double-limb jump landing task.

Testing sessions were conducted 7-10 days apart in order to ensure no carry-over effects from the intervention condition performed during the first testing session. Participants were

asked to maintain their physical activity and sport participation during this time. At the second testing session, the same aforementioned procedures were conducted, with the exception of the assignment for the intervention protocol; the participants performed the intervention condition that they did not perform during the first testing session.

Data Processing

Data Acquisition

Three-dimensional kinematic data were collected using a ten-camera motion capture system (Vicon Motion Systems, Centennial, CO, USA) sampled at 120Hz and filtered using a fourth-order low-pass Butterworth filter with a 20Hz cutoff frequency. Kinetic data were sampled at 1200Hz using two floor embedded force plates (Bertec Corporation, Columbus, OH, USA). Knee and ankle joint center coordinates were defined as the centroid between the medial and lateral condyles and malleoli identified during the static trail. Hip joint center coordinates were estimated from the coordinates of the L4-5, right ASIS, and left ASIS markers using the Bell method.[222] Reference frames for the foot, tibia, and femur were defined based on 3D-coordinates and segments as follows: 1st and 5th metatarsal heads, ankle joint center, and calcaneus (foot); medial and lateral malleoli, knee and ankle joint centers, and shank (tibia); medial and lateral femoral condyles, knee and hip joint centers, and thigh (femur). Joint angles were defined based on the position of the distal segment relative to the proximal segment using a Cardan angle sequence in the following order of rotation: sagittal (y-axis), frontal (x-axis), and transverse (z-axis).

Data Reduction

Marker coordinate and ground reaction force data was transferred into The Motion Monitor software (Innovative Sports Training, Chicago, Illinois) to build three-dimensional link-segment models for biomechanical data analysis and reduction. Lower extremity biomechanics for each limb were evaluated during the stance phase, which was defined as the interval from initial contact (IC) to toe-off.[223] IC was defined as the time point when the vertical ground reaction force (vGRF) exceeded 10N and toe-off as the time point when vGRF dropped below10N.[223]

Sagittal plane knee angle (°) was extracted at IC, peak, and displacement across the descending phase (peak knee flexion angle (°) – knee flexion angle at IC (°)). Ground reaction force data and processed segment data were used to calculate net internal sagittal and frontal plane knee and hip joint moments using inverse dynamics procedures.[146] Peak sagittal and frontal plane internal hip and knee moments and vGRF were extracted. Patellar tendon force (F_{PT}) was estimated using previously defined methodology of Nisell and Ekholm, through which F_{PT} is calculated by dividing the internal knee extension moment by the patellar tendon moment arm.[160] PT moment arms were calculated as a function of knee joint angles using the methodology of Herzog and Read.[144] Internal moments were normalized to the product of the participant's body weight and height ($N*m[kg*m]^{-1}$), while vGRF and F_{PT} was normalized to body weight (BW). Knee power (J/s) was calculated as the product of the internal sagittal plane knee moment ($N*m[kg*m]^{-1}$) * knee flexion velocity (°/millisecond).

F_{PT} impulse ($F_{PT}I$) and internal knee extension moment impulse (KEMI) were calculated as the area under the F_{PT} and KEM curves, respectively, during the descending phase of the landing task. Knee power (J/s) was calculated as the product of the internal sagittal plane knee

moment (Nm/kg*m) * knee flexion velocity (°/millisecond). Negative knee work (KW) (J) was calculated as the area negative under the knee power curve during the descending phase of the landing task.

Change scores for biomechanical dependent variables were calculated for each intervention condition as: change score (Δ) = $mean_{post} - mean_{pre}$.

STATISTICAL ANALYSIS

Demographic data was compared across groups using a one-way analysis of variance and Tukey post-hoc tests for pairwise comparisons. All pre- and post-intervention and change scores for biomechanical data were inspected for normality using the Shapiro-Wilk Test and normal Q-Q plots. We established four *a priori* comparisons to investigate both within-group and between-group effects of each intervention condition (isometric and sham-TENS). A mixed-model repeated measures ANOVA with 1 between (group: SYM, ASYM) and 1 within (intervention: isometric, sham-TENS) was conducted to compare change scores with an *a priori* alpha level of 0.05. Post hoc testing of significant group-by-intervention interactions was performed using *t*-tests with Bonferroni correction ($\alpha = 0.05/4 = 0.0125$) to account for pre-planned comparisons between groups for each intervention. Independent *t*-tests were used to compare between groups for each intervention and dependent *t*-tests were used to compared between interventions for each group. Cohen's *d* effect sizes were used to evaluate the magnitude of between group differences for loading volume variables, classified as weak ($d \leq 0.2$), small ($d = 0.2$-0.5), moderate ($d = 0.5$-0.8), or large ($d \geq 0.8$) (Table 4).[226] Due to group differences in several baseline biomechanical variables (manuscript 1), secondary analysis including a mixed-model repeated-measures analysis of covariance (ANCOVA) was conducted to compare change scores

for each biomechanical variable of interest while controlling for the baseline value for a given biomechanical variable. All statistical analyses were completed using SPSS v22 (IBM Inc., Armonk, New York, USA).

Table 1: Descriptive characteristics of the study population.

	Asymptomatic Tendinopathy (ASYM) (n=15)	**Symptomatic Tendinopathy (SYM) (n=13)**
Age (yrs)	21.13 ± 1.88	19.62 ± 1.61
Height (m)	1.84 ± 0.07	1.82 ± 0.05
Mass (kg)	81.45 ± 13.26	83.46 ± 5.12
Tegner Activity Scale (0-10)	7.93 ± 1.03	8.00 ± 1.00
Pubertal Development Scale (0-12)	11.87 ± 0.52	11.39 ±0.87
VISA-P (0-100)	94.40 ± 7.72	76.15 ± 13.37*

*statistically significant difference than ASYM group (p<0.001, MD: -18.25 (-26.41, -10.08))

Table 2: Single leg decline squat (SLDS) pain scores (NRS: 0-10) during each testing session.

		Asymptomatic Tendinopathy (ASYM) (n=15)	**Symptomatic Tendinopathy (SYM) (n=13)**
	Screening Session	0	3.23±1.21
Isometric Intervention Session	Pre-Landing Protocol	0	2.54±1.76
	Pre-Isometric Intervention	0.40±1.55	2.34±2.10
	Post-Isometric Intervention	0.60±1.68	1.62±1.89
	Isometric Intervention Change Score	0.20±0.77	-0.73±0.72
Sham-TENS Intervention Session	Pre-Landing Protocol	0.33±1.29	3.07±1.85
	Pre-Sham TENS Intervention	0.33±1.29	3.03±1.98
	Post-Sham TENS Intervention	0.33±1.29	2.42±1.63
	Sham-TENS Intervention Change Score	0	-0.62±1.12

RESULTS

The CONSORT diagram for recruitment and enrollment is detailed in Figure 1. Descriptive characteristics and pain scores are presented in Table 1 and Table 2, respectively. No significant differences in height, mass, or age were observed between groups ($p > 0.05$). The VISA-P score was significantly lower in the SYM group than the ASYM group ($p<0.001$), and the mean difference exceeded the MCID (13 points) for this subjective outcome measure (Table 1).[204] There were no group x intervention interactions for change in SLDS NRS pain ($F_{(1, 26)} = 0.555$, $p=0.463$) (Table 2). Group and individual participant pre- and post-intervention and change scores for NRS pain are visualized in Figures 2-5.

Descriptive characteristics for group and intervention condition are detailed in Table 3. For our within-group comparisons, there was one significant group x intervention interaction for VGRF ($F_{(1, 26)} = 5.33$, $p = 0.029$). However, post-hoc testing with Bonferroni correction ($\alpha = 0.05/4 = 0.0125$) demonstrated no statistical significance. Dependent-samples *t*-tests for each group demonstrated no statistical significance (ASYM: $t = -1.7$, $p = 0.107$; SYM: $t = -1.679$, $p = 0.119$). Independent *t*-tests demonstrated no statistical significance (isometric: $t = -2.58$, $p = 0.016$; sham-TENS: 0.72, $p = 0.460$). There were no further significant group x intervention interactions ($p>0.05$). Additionally, the ANCOVA analyses, including baseline biomechanical variables as co-variates, demonstrated the same outcome, as there were no significant group x intervention interactions observed ($p>0.05$).

Table 3: Descriptive characteristics (mean difference, standard deviation, 95% CI) for each biomechanical variable change score for the symptomatic and asymptomatic groups for the isometric and sham-TENS intervention conditions.

		Isometric Condition			**Sham-TENS Condition**			*Group x Condition*	
Biomechanical Variable	**Group**	***Mean* Δ**	***SD***	***95% CI***	***Mean* Δ**	***SD***	***95% CI***	**F**	***p-value***
Knee flexion angle @ IC (°)	SYM	-2.61	3.02	-4.44, -0.78	-1.67	3.15	-3.57, 0.24	1.56	0.223
	ASYM	-4.32	4.45	-6.78, -1.86	-1.57	4.14	-3.87, 0.72		
Peak knee flexion angle (°)	SYM	-3.58	4.91	-6.55, -0.61	-0.79	4.24	-3.35, 1.77	0	0.995
	ASYM	-4.36	7.45	-8.48, -0.23	-1.57	4.72	-4.19, 1.03		
Knee flexion displacement (°)	SYM	-0.97	5.99	-4.59, 2.64	0.88	3.97	-1.52, 3.27	0.44	0.511
	ASYM	-0.04	6.56	-3.67, 3.60	-0.01	4.82	-2.67, 2.65		
Peak VGRF (BW)	SYM	0.46	0.47	0.18, 0.75	0.21	0.23	0.08, 0.35	5.33	**0.029***
	ASYM	-0.04	0.54	-0.34, 0.26	0.32	0.50	0.04, 0.60		
Peak Internal KEM ($N*[kg*m]^{-1}$)	SYM	-0.007	0.26	-0.023, 0.009	0.007	0.03	-0.01, 0.02	2.41	0.132
	ASYM	0.005	0.01	-0.003, 0.01	0.002	0.02	-0.01, 0.02		
Peak F_{PT} (BW)	SYM	-0.14	0.43	-0.40, 0.12	0.20	0.46	-0.08, 0.47	4.06	0.054
	ASYM	0.13	0.32	-0.05, 0.31	0.10	0.38	-0.12, 0.31		
Peak knee power (J/s)	SYM	-0.07	0.38	-0.30, 0.16	-0.03	0.44	-0.29, 0.24	0.21	0.651
	ASYM	0.03	0.28	-0.13, 0.18	-0.02	0.38	-0.24, 0.19		
KEM impulse (Nm*ms)	SYM	-0.44	5.65	-3.85, 2.97	1.63	3.76	-0.64, 3.90	1.86	0.185
	ASYM	2.22	4.56	-0.30, 4.75	1.47	3.93	-0.71, 3.65		
F_{PT} impulse (BW*ms)	SYM	-6.84	123.19	-81.28, 67.60	35.29	80.96	-13.63, 84.21	1.70	0.204
	ASYM	51.13	102.60	-5.68, 107.95	33.94	87.62	-14.58, 82.47		
Negative knee work (J/kg)	SYM	-2.75	26.54	-18.78, 13.29	8.69	25.02	-6.43, 23.81	2.89	0.101
	ASYM	11.71	21.50	-0.20, 23.61	3.85	19.99	-7.22, 14.92		

Legend: Δ**:** change; IC: initial contact; VGRF: vertical ground reaction force; KEM: knee extension moment; F_{PT}: patellar tendon force

*statistically significant at $p < 0.05$.

Table 4: Cohen's *d* effect sizes for mean differences (pre-post) within each group for the isometric and sham-TENS intervention conditions.

		Isometric Condition	**Sham-TENS Condition**
Biomechanical Variable	**Group**	Cohen's *d*	Cohen's *d*
Knee flexion angle @ IC (°)	SYM	-0.37	-0.22
	ASYM	-0.92	-0.39
Peak knee flexion angle (°)	SYM	-0.30	-0.06
	ASYM	-0.38	-0.10
Knee flexion displacement (°)	SYM	-0.08	0.07
	ASYM	0.003	0.08
Peak VGRF (BW)	SYM	0.67	0.29
	ASYM	-0.04	0.50
Peak Internal KEM ($N*[kg*m]^{-1}$)	SYM	-0.25	0.24
	ASYM	0.18	0.09
Peak F_{PT} (BW)	SYM	-0.27	0.33
	ASYM	0.27	0.21
Peak knee power (J/s)	SYM	-0.14	-0.05
	ASYM	0.05	-0.03
KEM impulse (Nm*ms)	SYM	-0.06	0.23
	ASYM	0.37	0.18
F_{PT} impulse (BW*ms)	SYM	-0.05	0.23
	ASYM	0.39	0.19
Negative knee work (J/kg)	SYM	-0.07	0.22
	ASYM	0.40	0.09

Figure 1: Study CONSORT Diagram

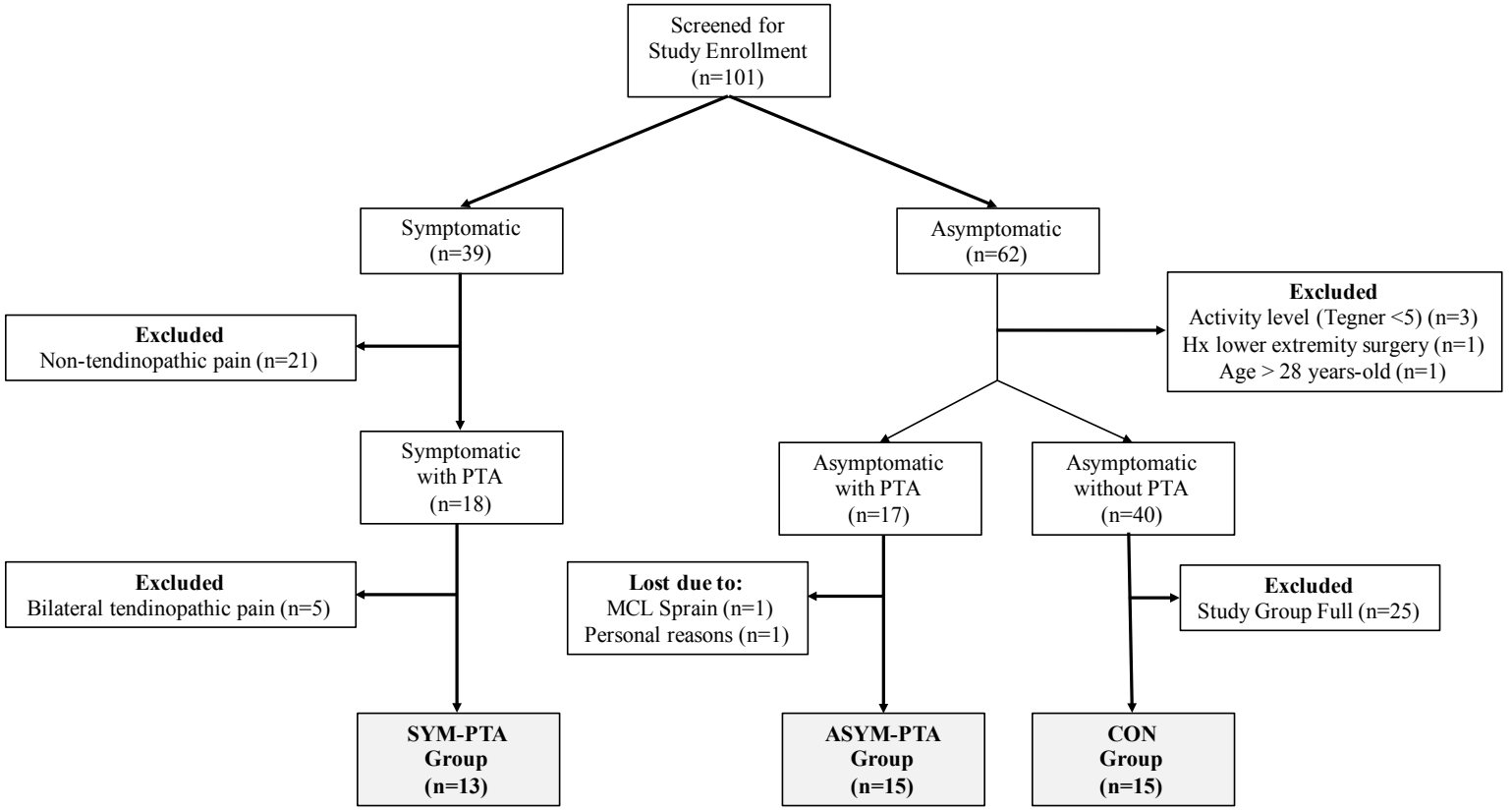


Figure 2: Individual participant SLDS pain (NRS: 0-10) change scores following the isometric (blue open circles) and sham-TENS (open red circles) interventions with median group change (black horizontal line).

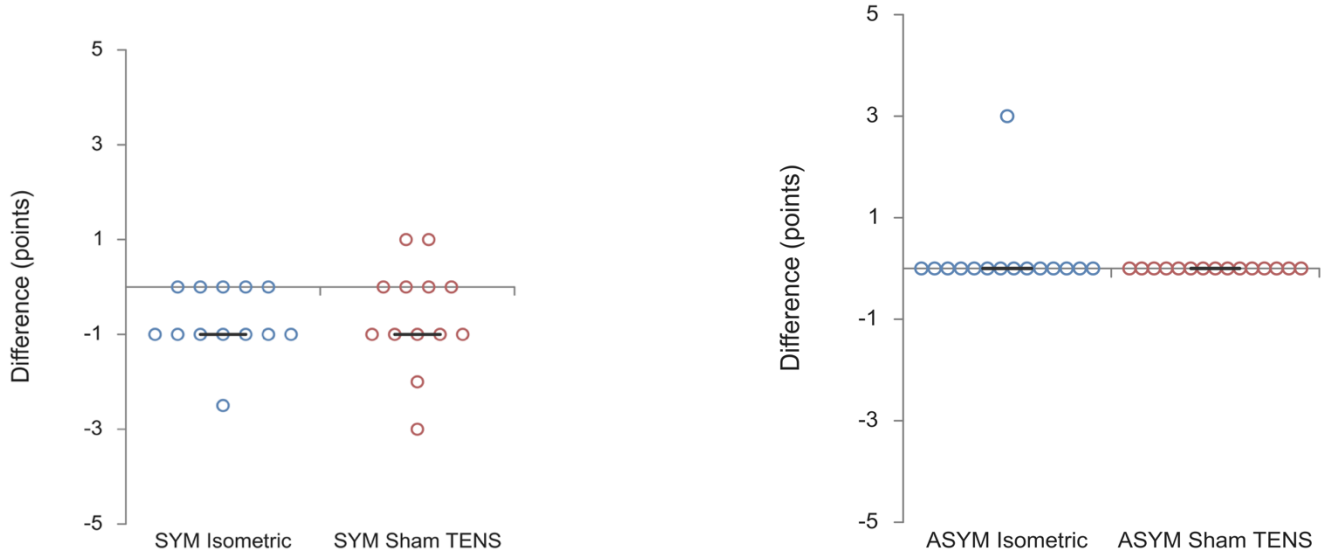


Figure 3: Mean and 95% confidence intervals for change scores for isometric and sham-TENS conditions for the SYM and ASYM groups.

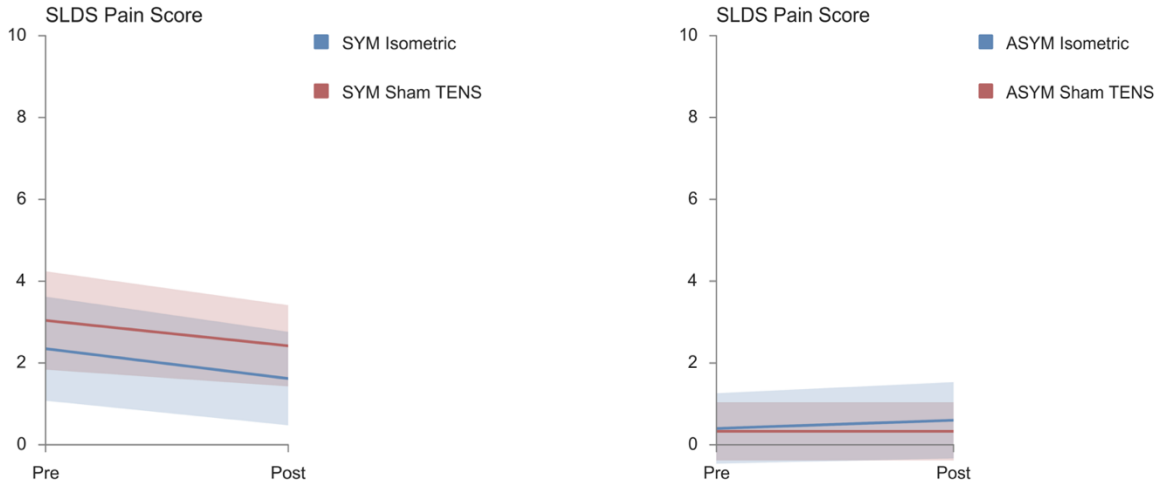
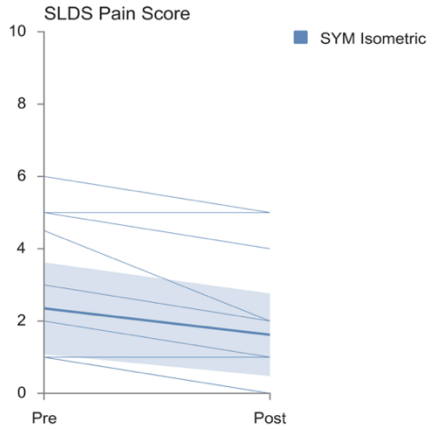
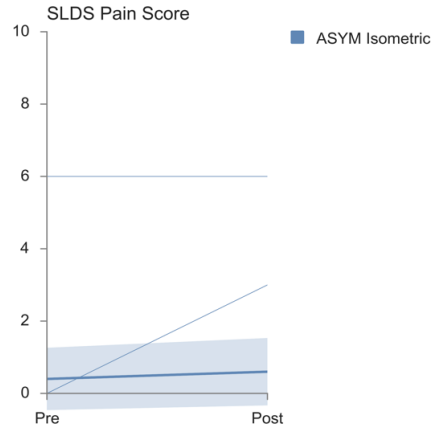


Figure 4: Individual participant pre- and post-isometric intervention SLDS pain scores (NRS 0-10) with mean (dark blue line) and 95% confidence bounds (shaded area)

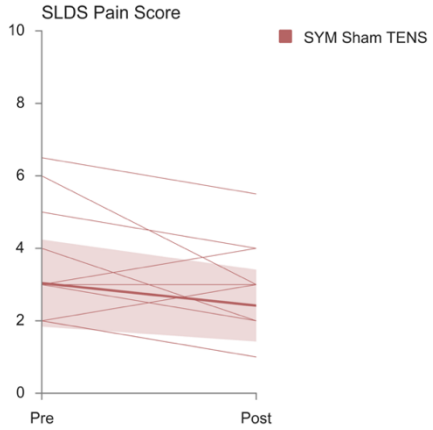


*Note: 2 participants reported pre- and post-NRS=0
2 participants reported pre- and post-NRS=1
3 participants reported pre-NRS=1 and post-NRS=0

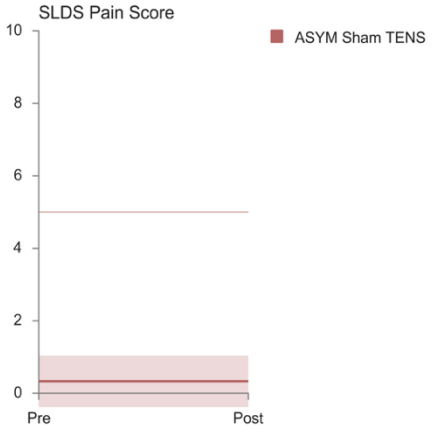


*Note: 13 participants reported pre- and post-NRS=0

Figure 5: Individual participant pre- and post-sham-TENS intervention SLDS pain scores (VAS 0-10) with mean (dark red line) and 95% confidence bounds (shaded area)



*Note: 2 participants reported pre-NRS=2 post-NRS=1
1 participant reported pre- and post-NRS=3
1 participant reported pre- and post-NRS =2



*Note: 14 participants reported pre- and post-NRS=0

DISCUSSION

We originally hypothesized that a single dose of an isometric loading intervention would result in reduced pain in the SYM group. As a result, we further hypothesized that the SYM group would demonstrate increased loading on the involved limb during the landing task, including increased FPT impulse, KEM impulse, negative knee work, and knee power. We did not expect to observe any other changes between conditions for either intervention group. In agreement with our hypotheses, there were no changes in biomechanics following the sham-TENS intervention in either group, and there was only one significant change in the ASYM group following the isometric intervention (reduced knee flexion angle at IC). Contrary to our hypothesis the SYM group did not demonstrate significant changes in pain or in knee loading biomechanics following the isometric intervention.

The SYM group has previously been shown to demonstrate a reduced biomechanical loading profile on the involved limb, including lesser knee flexion, patellar tendon force, and knee power in comparison to healthy control participants. Thus, there was the opportunity for increasing biomechanical loading in the SYM group following the isometric intervention. However, our results demonstrate weak to small effect of the isometric intervention in the SYM for our key variables of interest, specifically sagittal plane knee motion variables (*d*: -0.08 to -0.37), sagittal plane knee extension loading variables (*d*: -0.06 to -0.25), and patellar tendon loading variables (*d*: -0.05 to -0.27). Our results demonstrated a group x intervention condition interaction for VGRF, but post-hoc testing revealed no significant between- or within-group differences.

Furthermore, examining mean differences for these key biomechanical variables indicates few change scores that would be considered clinically meaningful. As noted in manuscript 1, the

SYM group demonstrated lesser knee flexion motion than the CON group for the majority of the stance phase at baseline testing (8-76%, *d*: 1.14±0.12, MD: 15.83±2.71°). However, there was only a mean change of 2.61° following the isometric intervention. So though the SYM group demonstrated a significantly different profile than the CON group on several key measures of knee loading, their profiles were not altered due to the intervention. Therefore, it appears that a single bout of isometric loading is not effective at changing landing biomechanics to increase loading magnitude during landing. There are several possible explanations for this finding.

The first consideration is the relatively lower baseline pain levels in the SYM group. Prior research by Rio et al. examining the same isometric intervention demonstrated significant immediate reductions in pain with large effects (*d*: 4.64) in adult male volleyball athletes (n=6).[17] In this study, the cohort presented with higher baseline pain (7.0/10 ± 2.04/10), considerably greater than in our cohort. Our SYM group demonstrated less pain prior to the isometric intervention (2.34/10 ± 2.10/10) and consequently displayed smaller effects from the isometric intervention (*d*: 0.36). In other research, van Ark et al (2015) showed reductions in pain and improvements in self-reported function in competing athletes following a four-week isometric loading intervention, but their baseline pain scores were also higher than our cohort (median: 6.3/10, IQR: 5.3-7/10).[16]

We are confident that we identified individuals with symptomatic patella tendinopathy as we used a rigorous, evidence-based inclusion criteria battery (SLDS pain ≥ 2/10 and ultrasound evidence of PTA). All athletes in the SYM group met our inclusion criteria of at least 2/10 SLDS at the screening session (mean±sd: 3.23±1.21) and also demonstrated reduced VISA-P scores (76.15 ± 13.37). However, on average, our cohort was on the low end of the pain spectrum compared to prior research. Therefore, we suspect that due to the low-levels of pre-intervention

pain in our cohort, there was not adequate room for a change in symptoms following the isometric loading intervention.

It is common for tendon pain to fluctuate across activities or time periods, particularly in competing athletes, which contributes to the challenge of managing this condition.[9,90] All athletes in the SYM group met our inclusion criterion of at least 2/10 SLDS at the screening session (mean ± sd: 3.23±1.21). A numeric rating scale (NRS) change score of 2 points has been associated with the concept of "much better" improvement of pain intensity in patients with chronic musculoskeletal pain, and is considered an appropriate cut-point for minimal clinically important difference (MCID) in pain.[206] The screening session occurred on a separate day approximately one week prior to the first intervention session. However, there were no differences in screening and pre-isometric (mean difference: 0.88±1.78 points, p=0.099) or pre-sham-TENS (mean difference: 0.19±1.75 points, p=0.699) pain scores.

Importantly, no SYM participants reported an increase in pain following the intervention (Figure 2) and while there was an average decrease in pain post-intervention (pre: 2.34 ± 2.10, post: 1.62 ± 1.89, mean change: -0.73 ± 0.72) (Figure 4), the magnitude of pain reduction did not meet the MCID of 2 points.[206] We performed a secondary analysis to determine associations between pre-intervention pain and change in pain in the symptomatic group around the isometric intervention, and these variables were not associated (r = -0.437, p = 0.136). This suggests that in our cohort, pre-intervention pain level did not associate with response to isometric intervention, likely attributable to the large range of pre-intervention pain scores (0-6). Additionally, we used a rigorous, evidence-based inclusion criteria battery (SLDS pain ≥ 2/10 and ultrasound evidence of PTA) for the SYM group. With further consideration of the SYM group's VISA-P score (76.15 ± 13.37), we feel confident that we correctly identified individuals with symptomatic

patellar tendinopathy. However, on average, our cohort was on the low end of the pain spectrum. Therefore, we suspect that due to the low-levels of pre-intervention pain in our cohort, there was not an adequate opportunity for a change in symptoms that may have influenced biomechanics in our cohort.

The second key consideration is that a single intervention may not be sufficient to change movement profiles in individuals with PT. Due to the cross-sectional nature of this study, we cannot determine the onset of structural abnormalities and/or pain in either group, which may influence how rooted an individual's potentially-altered movement profile may be. Previous research has demonstrated a pattern of under-loading in athletes with symptomatic PT.[19,21] Additionally, as discussed in manuscripts 1 and 2, our SYM cohort demonstrated reduced loading on the involved limb compared to healthy controls, including less knee flexion motion, patellar tendon force, and energy absorption supported by large effect sizes, while our ASYM cohort did not demonstrate patterns of overloading. Thus, the SYM cohort had room for improvement in their involved limb loading.

These findings reinforce that the presence of symptoms is a key factor that influences reduced magnitude of loading during landing tasks in those with PTA. We hypothesized that, regardless of duration of symptoms, the isometric protocol would invoke an improved willingness to load during the landing task, as isometrics have been shown to acutely reduce cortical inhibition without a decline in quadriceps muscle performance.[17] The ability to increase biomechanical load magnitude during functional tasks may be an important aspect of interventions shown to reduce pain. By modifying symptomatic participants' biomechanical load profiles during landing, there is the potential to augment the amount of tendon loading and ultimately further facilitate positive mechano-therapeutic effects on tendon capacity. However,

we did not observe these changes in biomechanical loading magnitude following a single bout of isometric loading in either the SYM or ASYM group. It is possible that multiple sessions of the isometric protocol over time may be necessary to realize the potential of this intervention to change biomechanics.

Additionally, there were no differences between the SYM and ASYM groups in the response to the isometric intervention. We hypothesized that the larger effects of the isometric protocol would be seen in the SYM group due to the presence of pain and the analgesic effect of the intervention. However, as discussed previously, the relatively low levels of baseline pain in the SYM group resulted in little room for clinically significant pain reduction. This ceiling effect in the SYM group may be responsible for the lack of group differences noted due to the isometric intervention.

The lack of significant findings in the current study highlights the need for future studies investigating the effects of isometric loading on landing biomechanics in athletes with higher baseline pain levels, as well as accounting for duration of symptoms. Future research may also consider investigating which modes of intervention are most effective, as it is possible that a load-based intervention plus verbal- and/or visual-feedback on important features of landing may be provide both the mechano-transductive and motor learning input needed to invoke change in movement. Finally, studying the effects of a longer duration isometric intervention (i.e. 3-4x/week for 4 weeks) on landing biomechanics may also have helpful clinical implications for rehabilitation protocols. While this study design was novel in terms of including three distinct groups, the magnitude of baseline pain in our symptomatic cohort may not have been adequate to elicit analgesia as previously demonstrated,[16,17] limiting the potential for influencing movement profiles.

LIMITATIONS

We acknowledge several limitations to this study. Though having reported moderate levels of activity related disability on the VISA-P, our symptomatic cohort was relatively functional, with only low to moderate pain levels reported before each intervention condition. While robust inclusion criteria were utilized to define our tendinopathy groups, we did not account for duration of symptoms in the SYM group, which may have influenced how long an individual may have developed and instilled aberrant movement profiles in response to persistent pain. Participants with greater pain may portray a different response to the isometric intervention than our cohort.

Additionally, the cross-sectional design of this study prevents us from determining whether the observed movement profiles were present prior to the development of PTA with or without symptoms, so we are unable to determine a cause-and-effect mechanism between biomechanics and patellar tendinopathy. While our estimation of patellar tendon force was chosen based on accepted models,[23,144,158,160] it is possible that these models underestimate the actual force acting through tendon. Finally, this study was conducted only on college-aged males so its findings cannot be extrapolated to females or males of a different age range.

CONCLUSIONS

The results of this study demonstrate that an isometric patellar tendon loading exercise protocol did not have acute effects on landing biomechanics in male athletes with symptomatic or asymptomatic tendinopathy. Though isometric tendon loading is a tolerable and analgesic treatment option for athletes with symptomatic patellar tendinopathy,[16–18] our findings suggest

that patient selection and duration of intervention implementation may be important factors if using isometric exercise to influence movement profiles.

Future research should examine the effects of a longer-duration isometric exercise intervention program on athletes with higher tendon pain and associated disability.

APPENDICES

Appendix 1. Pubertal Development Scale

Five Question Method

Questions

1. "Would you say that your growth in height: __"
2. "Would you say that your body hair growth: __" (body hair means anywhere other than your head.
3. "Have you noticed any skin changes, especially pimples: __"

FOR BOYS:

4. "Have you noticed deepening of your voice?"
5. "Have you begun to grow hair on your face?"

FOR GIRLS:

4. "Have you noticed that your breasts have begun to grow?"

5a. "Have you begun to menstruate (started to have your period)?"

5b. "If yes, how old were you when you started to menstruate)?"

Scoring Criteria

Items 1-4 on girls' version and all items on the boys' version will be provided with response options of:

1 point = not started
2 points = barely started
3 points = definitely underway
4 points= seems completed

Items 5 on the girls' version will be scored as:

1 point = no
4 points = yes

Pubertal Stages

Stage 1: PDS Score = 3: pre-pubertal
Stage 2: PDS Score = 4 or 5: pubertal
Stage 3: PDS Score = 6, 7, or 8: pubertal
Stage 4: PDS Score = 9, 10, 11: pubertal
Stage 5: PDS Score = 12: puberty complete

Appendix 2. Tegner Activity Level Scale.

TEGNER ACTIVITY LEVEL SCALE

Please indicate in the spaces below the HIGHEST level of activity that you participated in BEFORE YOUR INJURY and the highest level you are able to participate in CURRENTLY.

BEFORE INJURY: Level_____ CURRENT: Level_____

Level 10	**Competitive sports- soccer, football, rugby (national elite)**
Level 9	**Competitive sports- soccer, football, rugby (lower divisions), ice hockey, wrestling, gymnastics, basketball**
Level 8	**Competitive sports- racquetball or bandy, squash or badminton, track and field athletics (jumping, etc.), down-hill skiing**
Level 7	**Competitive sports- tennis, running, motorcars speedway, handball** **Recreational sports- soccer, football, rugby, bandy, ice hockey, basketball, squash, racquetball, running**
Level 6	**Recreational sports- tennis and badminton, handball, racquetball, down-hill skiing, jogging at least 5 times per week**
Level 5	**Work- heavy labor (construction, etc.)** **Competitive sports- cycling, cross-country skiing,** **Recreational sports- jogging on uneven ground at least twice weekly**
Level 4	**Work- moderately heavy labor (e.g. truck driving, etc.)**
Level 3	**Work- light labor (nursing, etc.)**
Level 2	**Work- light labor** **Walking on uneven ground possible, but impossible to back pack or hike**
Level 1	**Work- sedentary (secretarial, etc.)**
Level 0	**Sick leave or disability pension because of knee problems**

Y Tegner and J Lysolm. *Rating Systems in the Evaluation of Knee Ligament Injuries.* Clinical Orthopedics and Related Research. Vol. 198: 43-49, 1985.

Appendix 3. International Physical Activity Questionnaire (IPAQ)

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?

   _____ days per week

   ☐ No vigorous physical activities → *Skip to question 3*

2. How much time did you usually spend doing vigorous physical activities on one of those days?

   _____ hours per day

   _____ minutes per day

   ☐ Don't know/Not sure

Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

   _____ days per week

   ☐ No moderate physical activities → *Skip to question 5*

4. How much time did you usually spend doing moderate physical activities on one of those days?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

_____ days per week

☐ No walking → *Skip to question 7*

6. How much time did you usually spend walking on one of those days?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

The last question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the last 7 days, how much time did you spend sitting on a week day?

_____ hours per day

_____ minutes per day

☐ Don't know/Not sure

This is the end of the questionnaire, thank you for participating.

Appendix 4. Knee Injury History Form.

Subject Number _____ Date _____

Please Circle (Yes or No) regarding your situation.

Yes	No	Have you had an injury to either leg (other than patellar tendinopathy) that has altered you function in the past 6 months?
Yes	No	Have you had a surgery to either leg (knee, ankle, hip) in the past 1 year?
Yes	No	Have you had an injection (corticosteroids, plasma-rich-protein, etc.) to the patellar tendon in the last 3 months?
Yes	No	Do you have any knee ligaments that have not been reconstructed?
Yes	No	Do you have any nerve injuries in your legs or lower back?
Yes	No	Do you have any known muscular abnormalities?
Yes	No	Do you have a heart condition that would stop you from exercising?
Yes	No	Have you ever been diagnosed with cancer over your knee or thigh?
Yes	No	Do you currently have an infection over your thigh or in your knee?
Yes	No	Do you know of a hypersensitivity to electrical stimulation?

1. Have you ever had a knee injury?

When (month / year): _____
Explain:_____
_____
_____

2. Have you ever had a knee surgery?

When (month / year): _____
Explain:_____
_____
_____

3. Have you participated in formal rehabilitation (physical therapy) for patellar tendinopathy in the last 3 months?

When did you start (month / year)? _____
For How Long? _____

4. Have you ever had an injury/surgery to your ankle, hip or lower back?

When (month/ year): _____
Explain:
_____
_____
_____

Appendix 5. Percentage of predicted mature height calculation

Predicted % of mature height = $\frac{mother's\ height + father's\ height}{2}$ x 100

(Malina et al, 2007)[263]

Appendix 6. Victorian Institute of Sport Assessment-Patellar Tendon questionnaire.

VICTORIAN INSTITUTE OF SPORT

1. For how many minutes can you sit pain free?

0 mins ☐☐☐☐☐☐☐☐☐☐☐ 100 mins Points ☐

0 1 2 3 4 5 6 7 8 9 10

2. Do you have pain walking downstairs with a normal gait cycle?

strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

3. Do you have pain at the knee with full active non-weightbearing knee extension?

strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain when doing a full weight bearing lunge?

strong severe pain ☐☐☐☐☐☐☐☐☐☐☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

5. Do you have problems squatting?

Unable ☐☐☐☐☐☐☐☐☐☐☐ no problems Points ☐

0 1 2 3 4 5 6 7 8 9 10

6. Do you have pain during or immediately after doing 10 single leg hops?

strong severe pain/unable ☐☐☐☐☐☐☐☐☐☐☐ no pain Points ☐

0 1 2 3 4 5 6 7 8 9 10

7. Are you currently undertaking sport or other physical activity?

0 ❒ Not at all

4 ❒ Modified training ± modified competition

7 ❒ Full training ± competition but not at same level as when symptoms began

10 ❒ Competing at the same or higher level as when symptoms began

8. Please complete **EITHER A, B or C** in this question.

• If you have **no pain** while undertaking sport please complete **Q8a only**.

• If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only**.

• If you have **pain that stops you from completing sporting activities**, please complete **Q8c only**.

8a. If you have **no pain** while undertaking sport, for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❒	❒	❒	❒	❒	Points ☐
0	7	14	21	30	

OR

8b. If you have some pain while undertaking sport, but it does not stop you from completing your training/practice for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❒	❒	❒	❒	❒	
0	4	10	14	20	Points ☐

OR

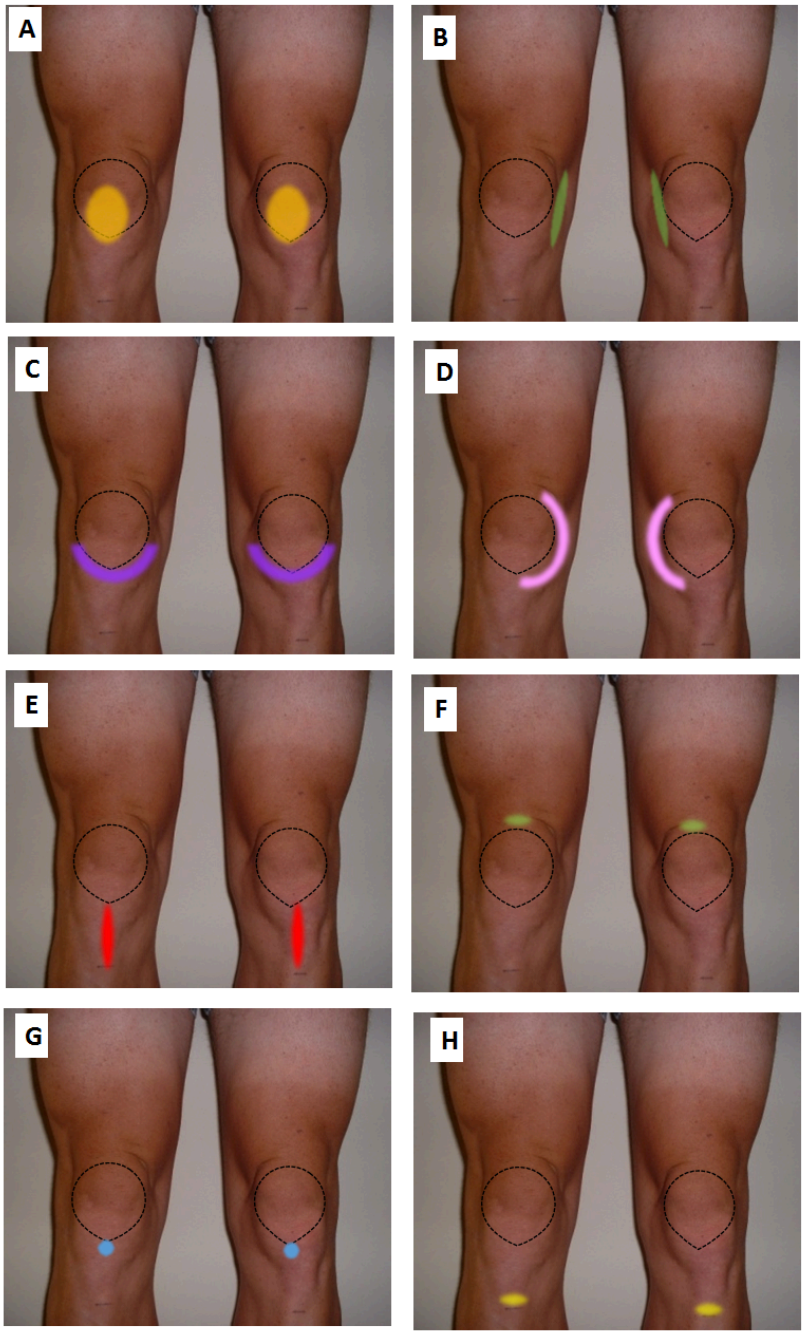
8c. If you have **pain which stops you** from completing your training/practice for how long can you train/practise?

NIL	1-5 mins	6-10 mins	7-15 mins	>15 mins	
❒	❒	❒	❒	❒	
0	2	5	7	10	Points ☐

TOTAL VISA SCORE ❒

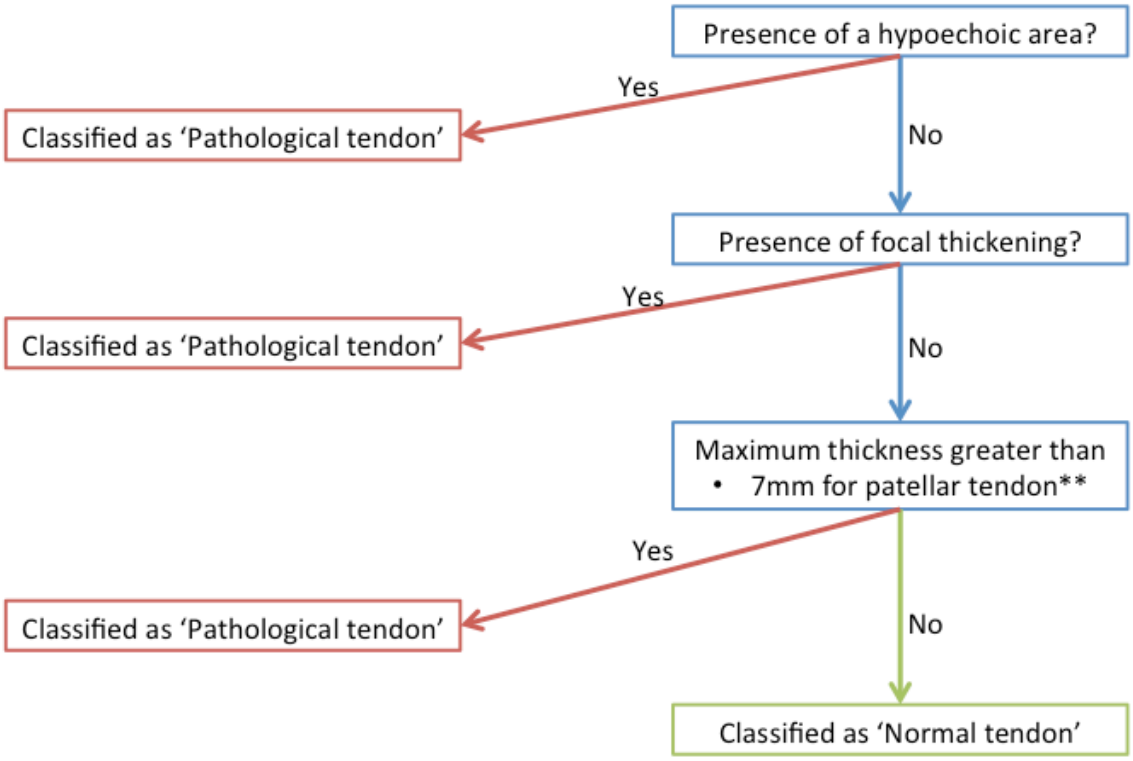
Appendix 7. Pain map.
Utilized for participant to determine location of pain immediately following single-limb decline squat. Selections 'E' and 'G' will be considered (+) for patellar tendon pain and will meet study inclusion criteria.

Please note the black line is an approximation of the kneecap.



Appendix 8. Algorithm for patellar tendon abnormality diagnosis.

Criteria decision-making in current study to characterize patellar tendon structural pathology using conventional ultrasound imaging (from Docking and Cook, 2015).[80]



**Schmid et al (2002). Is impingement the cause of jumper's knee? Dynamic and static magnetic resonance imaging of patellar tendinitis in an open-configuration system. Am J Sports Med

Appendix 9. ActiGraph Wear Position

Appendix 10. Supplementary material for manuscript 3.

Manuscript 3: Secondary Analysis

The following data reflect a secondary analysis, by which 4 total participants were removed from the sample:

- ASYM (n=1) due to PFPS reported at the pre- and post-intervention (6/10 isometric, 5/10 sham-TENS) time points (after having 0/10 pain at both screening and at pre-landing time points)
- SYM (n=3) due to not meeting the screening inclusion criteria of at least 2/10 pain at the pre-landing time point.
- This results in samples of ASYM: n=14 and SYM: n=10.

Table 1: Descriptive characteristics of the study population.

	Asymptomatic Tendinopathy (n=14)	**Symptomatic Tendinopathy (n=10)**
Age (yrs)	21.00±1.88	19.50±1.78
Height (m)	1.84±0.07	1.83±0.03
Mass (kg)	81.54±13.76	83.18±5.17
Tegner Activity Scale (0-10)	8.00±1.04	7.90±0.99
Pubertal Development Scale (0-12)	11.86±0.53	11.40±0.84
VISA-P (0-100)	95.57±6.48	74.80±14.13*

*statistically significant difference than ASYM group (p<0.001, MD: -20.77 (11.91, 29.63))

Table 2: Single leg decline squat (SLDS) pain scores (NRS: 0-10) during each testing session.

		Asymptomatic Tendinopathy (n=14)	Symptomatic Tendinopathy (n=10)
	Screening Session	0	4.2±0.92
Isometric Intervention Session	Pre-Landing Protocol	0	3.20±1.40
	Pre-Isometric Intervention	0	2.95±2.01
	Post-Isometric Intervention	0.21±0.80	2.00±2.00
	Isometric Intervention Change Score	+0.21±0.80	-0.95±0.69
Sham-TENS Intervention Session	Pre-Landing Protocol	0	3.20±1.75
	Pre-Sham TENS Intervention	0	3.35±2.03
	Post-Sham TENS Intervention	0	2.65±1.67
	Sham-TENS Intervention Change Score	0	-0.70±1.25

Table 3: Descriptive characteristics (mean difference, standard deviation, 95% CI) for each biomechanical variable change score for the symptomatic and asymptomatic groups for the isometric and sham-TENS intervention conditions.

		Isometric Condition			**Sham-TENS Condition**			*Group x Condition*	
Biomechanical Variable	**Group**	***Mean* Δ**	***SD***	***95% CI***	***Mean* Δ**	***SD***	***95% CI***	***F***	***p*-value**
Knee flexion angle @ IC (°)	SYM	-2.21	3.30	-4.57, 0.15	-0.91	2.90	-2.98, 1.16	0.615	0.441
	ASYM	-3.65	3.74	-5.80, -1.49	-1.07	3.79	-3.25, 1.12		
Peak knee flexion angle (°)	SYM	-3.57	4.08	-6.49, -0.66	-0.53	4.33	-3.64, 2.57	0.038	0.847
	ASYM	-4.05	7.63	-8.46, 0.36	-1.57	4.90	-4.40, 1.25		
Knee flexion displacement (°)	SYM	-1.37	5.96	-5.63, 2.90	0.38	4.38	-2.75, 3.51	0.358	0.556
	ASYM	-0.40	6.65	-4.24, 3.43	-0.51	4.58	-3.15, 2.34		
Peak VGRF (BW)	SYM	0.51	0.53	0.13, 0.89	0.26	0.24	0.08, 0.43	4.441	**0.047***
	ASYM	-0.03	0.56	-0.36, 0.29	0.36	0.50	0.08, 0.65		
Peak Internal KEM ($N*[kg*m]^{-1}$)	SYM	-0.007	0.03	-0.03, 0.01	0.008	0.02	-0.01, 0.02	1.882	0.184
	ASYM	0.002	0.01	-0.004, 0.009	0.001	0.02	-0.01, 0.02		
Peak F_{PT} (BW)	SYM	-0.14	0.49	-0.49, 0.21	0.18	0.40	-0.11, 0.46	2.513	0.127
	ASYM	0.07	0.24	-0.06, 0.21	0.08	0.40	-0.15, 0.31		
Peak knee power (J/s)	SYM	-0.063	0.43	-0.37, 0.24	0.01	0.32	-0.22, 0.25	0.278	0.603
	ASYM	0.002	0.28	-0.16, 0.16	-0.03	0.40	-0.26, 0.19		
KEM impulse (Nm*ms)	SYM	0.60	4.83	-2.86, 4.05	1.35	3.75	-1.34, 4.03	0.313	0.581
	ASYM	1.60	4.02	-0.73, 3.92	1.44	4.08	-0.92, 3.79		
F_{PT} impulse (BW*ms)	SYM	16.73	103.14	-57.06, 90.52	29.03	79.57	-27.89, 85.96	0.224	0.641
	ASYM	37.52	91.33	-15.22, 90.25	33.20	90.88	-19.27, 85.67		
Negative knee work (J/kg)	SYM	-4.51	21.67	-20.01, 11.00	6.81	26.67	-12.27, 25.89	2.442	0.132
	ASYM	9.16	19.82	-2.28, 20.61	3.79	20.74	-8.18, 15.77		

Legend: Δ**:** change; IC: initial contact; VGRF: vertical ground reaction force; KEM: knee extension moment; F_{PT}: patellar tendon force

*statistically significant at $p < 0.05$.

Table 4: Cohen's *d* effect sizes for mean differences (pre-post) within each group for the isometric and sham-TENS intervention conditions.

		Isometric Condition	**Sham-TENS Condition**
Biomechanical Variable	**Group**	Cohen's *d*	Cohen's *d*
Knee flexion angle @ IC (°)	SYM	-0.29	-0.11
	ASYM	-0.78	-0.34
Peak knee flexion angle (°)	SYM	-0.34	-0.04
	ASYM	-0.35	-0.10
Knee flexion displacement (°)	SYM	-0.13	0.03
	ASYM	-0.03	0.04
Peak VGRF (BW)	SYM	0.79	0.37
	ASYM	-0.04	0.56
Peak Internal KEM (N*[kg*m]$^{-1}$)	SYM	-0.27	0.24
	ASYM	0.10	0.07
Peak F_{PT} (BW)	SYM	-0.28	0.27
	ASYM	0.16	0.19
Peak knee power (J/s)	SYM	-0.13	0.03
	ASYM	0.005	-0.05
KEM impulse (Nm*ms)	SYM	0.09	0.19
	ASYM	0.27	0.17
F_{PT} impulse (BW*ms)	SYM	0.12	0.19
	ASYM	0.29	0.18
Negative knee work (J/kg)	SYM	-0.12	0.17
	ASYM	0.32	0.08

Summary of Results of Secondary Analysis

A mixed-model repeated measures ANOVA with 1 between (group: SYM, ASYM) and 1 within (intervention: isometric, sham-TENS) was conducted to compare change scores with an *a priori* alpha level of 0.05. Post hoc testing of significant group-by-intervention interactions was performed using *t*-tests with Bonferroni correction ($\alpha = 0.05/4 = 0.0125$) to account for pre-planned comparisons between groups for each intervention. Independent *t*-tests were used to compare between groups for each intervention and dependent *t*-tests were used to compared between interventions for each group.

From this analysis, there was one significant group x intervention interaction for peak VGRF ($F_{(1, 22)} = 4.441$, $p = 0.047$). Dependent-samples *t*-tests for each group demonstrated no statistical significance (ASYM: $t = -1.8$, $p = 0.095$; SYM: $t = -1.317$, $p = 0.22$). Independent *t*-tests demonstrated no statistical significance (isometric: $t = -2.4$, $p = 0.025$; sham-TENS: $t = 0.683$, $p = 0.503$). Therefore, the findings of this secondary analysis demonstrate that there were no statistically significant effects of the isometric intervention in either group. Removing the individuals that no longer met screening level criteria from the analysis did not change our original findings.

Figure 1: Individual participant SLDS pain (NRS: 0-10) change scores following the isometric and sham-TENS interventions for each group median group change (black horizontal line).

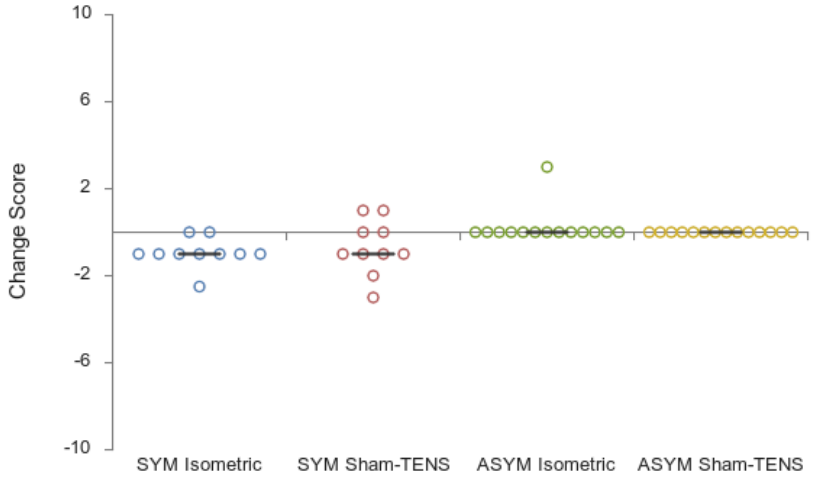


Figure 2: Individual participant pre- and post-isometric intervention SLDS pain scores (NRS: 0-10) with mean (dark line) and 95% confidence bounds (shaded area)

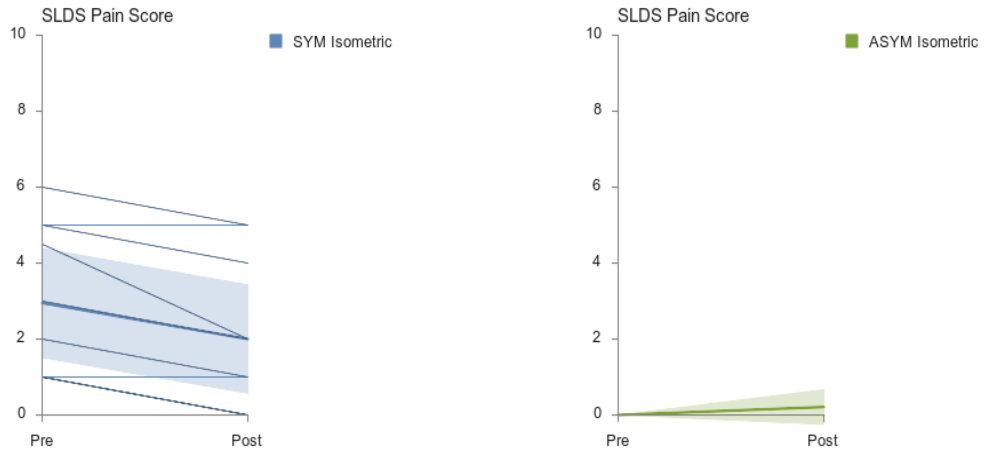
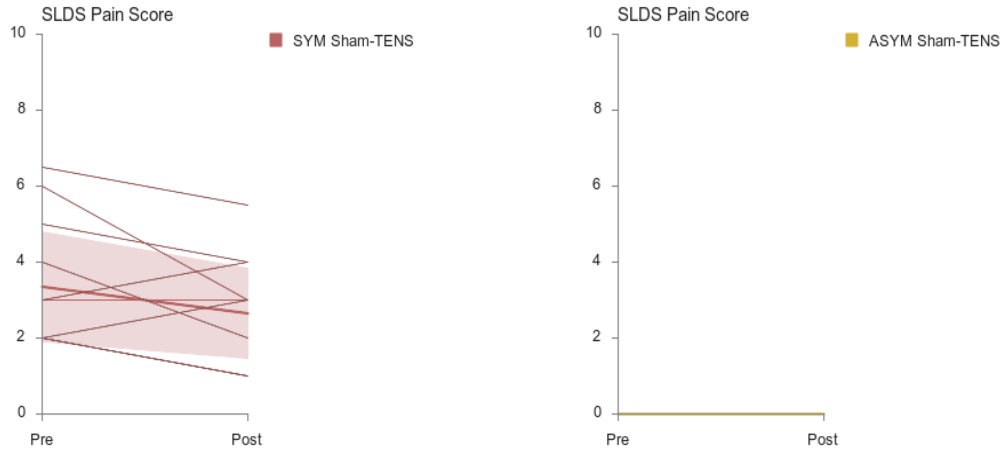


Figure 3: Figure 2: Individual participant pre- and post-sham-TENS intervention SLDS pain scores (NRS: 0-10) with mean (dark line) and 95% confidence bounds (shaded area).



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